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Walter A. Jillson.

A TEXT-BOOK
OF
CLINICAL MEDICINE.

PRINCIPLES OF DIAGNOSIS.

BY CLARENCE BARTLETT, M.D.,
PROFESSOR OF CLINICAL MEDICINE AND ASSOCIATE PROFESSOR OF MEDICINE IN THE HAHNEMANN MEDICAL COLLEGE OF PHILADELPHIA; SENIOR NEUROLOGIST
TO THE HAHNEMANN HOSPITAL, ETC.

WITH 245 ILLUSTRATIONS, INCLUDING SIX COLORED PLATES.

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PREFACE.

In presenting a review of practical medicine, one may proceed in one of two ways. He may adopt the method in vogue for many years, that of describing the clinical pictures of the various diseases, presenting details as to their etiology, symptomatology, diagnosis, prognosis and treatment. Or, he may approach the subject from the opposite standpoint, describing the various symptoms which go to demonstrate that a person is ill, and give details as to their clinical relations and diagnostic significance. Inasmuch as the latter plan follows the lines of argument one employs when examining a case, it may be called the clinical method.

The old or established system of teaching medicine has its advantages; otherwise it would not have preserved its popularity even unto the present time. But it has its disadvantages, which become more and more obvious as we have forced upon us the truth of the axiom, "We must treat the patient, not his disease." To label a given case of illness with the name of one or the other diseases described in the standard works on medicine is not making a diagnosis, because the standard clinical types as described in such works are the exception, and not the rule. We must, therefore, be able to proceed still further in our conclusion, and obtain an adequate conception of all the pathological changes taking place in the patient in whose case we happen to be interested, even though we are unable to present our conclusion in the shape of some set title. All of these associated changes cannot be studied practically according to the old system of expounding the principles of medical practice.

Approaching the subject from the reverse side,—the clinical,—the study of cases becomes interesting and easier. The problem is solved, not by matching the case in hand with the type as described in a standard text-book, but by a process of reasoning. This method of study is, in reality, the only one possible in unravelling the mysteries of the majority of cases of chronic disease.

The present work has been in mind a number of years, and was prompted by the above thoughts.

Another advantage in presenting a study of medicine according to the clinical method is that one finds greater opportunities for describing methods of treatment. Every one is well aware that the treatment of some one disease *per se* takes but a few words to describe,—and even these few words are unsatisfactory,—and yet it is possible to say much concerning the treatment and management of patients who are suffering with that disease. So, when the present work was planned, the author decided to include chapters on therapeutics based upon those devoted to diagnosis. It was quite a problem with him, at first, to decide how the subject-matter should be arranged,—*i.e.*, whether he should follow each diagnostic chapter with appropriate therapeutic remarks, or have one volume devoted entirely to diagnosis, the other to treatment. The latter course was finally decided to be the better, as it makes the work easier for reference and consecutive reading, and each volume becomes complete in itself.

In some few places, *e.g.*, the paragraphs devoted to electro-diagnosis, the author has taken the liberty of quoting his previous writings, as time has made no changes in our knowledge respecting said subjects.

In closing, the author desires to express his appreciation of the kindly criticisms of a number of his colleagues, especially those of Drs. W. B. Van Lennep, C. M. Thomas, W. C. Goodno, and H. S. Weaver, and his indebtedness to others who assisted in the preparation of certain chapters, and whose names are appended thereto. Valuable assistance has also been afforded by Drs. F. Mortimer Lawrence, John J. Tuller, and G. M. Golden.

To Dr. W. H. Lyle he is indebted for a number of drawings, as well as for the excellent colored plates representing the appearance of dried and stained blood-specimens.

He also takes pleasure in acknowledging the courtesy of P. Blakiston's Son & Co. for the use of the colored plates illustrating the various phases of the malarial parasites.

CLARENCE BARTLETT, M.D.

1506 ARCH STREET, PHILADELPHIA.
November 15, 1902.

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ERRATUM.

P. 579, line 4. For "hydronephrosis," read "pyonephrosis."

CLINICAL MEDICINE.

PRINCIPLES OF DIAGNOSIS.

CHAPTER I.

THE EXAMINATION OF PATIENTS.

"More mistakes are made by not looking than by not knowing." In other words, if proper attention is given to the thorough examination of patients, fewer mistakes will be charged up against the medical profession. Almost any one is capable of giving a correct diagnosis, and of outlining a successful plan of treatment, if the complete data of the case are presented to him. On the other hand, the number of men who take the time and trouble to examine their patients properly is comparatively small. A certain part of this carelessness is due to the patients themselves, in that they affect a mock modesty. Many times have I heard physicians say "I wish I could take the time to make thorough examinations, but the people will not pay for it;" or, "If we were to make it a rule to go carefully over each case, the people would get tired of us;" or still again, "The people resent such close questioning." Replies to these objections are easily made. "What is worth doing, is worth doing well." If one does not examine his cases thoroughly, he can depend upon it that the day is not far distant when he will become fossilized. As a mere routinist, he grows into an old man while still young in years. He thus becomes incapacitated for competition with the young and rising generation. If, on the other hand, he makes it a rule to conduct complete and systematic examinations, he will find that, with practice, such methods are not as time-consuming as would at first sight appear. Many of the data, which are obtainable by the consulting physician only after a painstaking investigation, are matters of observation from everyday contact with the family physician. As to people objecting to thorough investigations of their illnesses, that is a mere fiction of a timid imagination. Patients who are that cranky had better be permitted to place themselves under the care of a less conscientious competitor. As to examinations eliciting matters discreditable to the patient, these objections would not hold, if physicians made it an inviolate rule to respect professional confidences. The writer believes that the number of physicians who are regularly careless is infinitesimal as compared with those who attempt good systematic work ; but it must be remembered that the derelictions of the bad one will be utilized by the public to degrade the ninety and nine.

As to methods to be pursued in making an examination, physicians will differ. Each follows his own favorite plan. As long as that plan is according to a definite system, it is good ; *but it must be systematic.* Nothing makes a physician so ridiculous in the eyes of his patient as unsystematic, aimless questioning and investigations.

The first question is an all-important one. Too many are inclined to start the interview with "What is the matter with you?" and with remarkable frequency comes the reply, "That is just what I want you to tell me." The more knowing ones actually hazard a diagnosis, and are inclined to doubt the skill of a physician who proves the same to be wrong. Instead of asking such a question, some commonplace words, as "Well !" or "Tell me about your illness," are sufficient to start the patient with his history. In this narrative it is not wise to interrupt him at first, as by permitting him to go his own way, even though he be illogical and verbose, one can discover considerable concerning his temperament and mental state. Only if he is inclined to wander too far from matters relevant to his illness should he be interrupted. The patient's story completed, then it becomes the examiner's duty to ask questions to clear up certain vague statements made in the history, or to determine what the patient had in mind when he made use of certain terms. For example, such terms as rheumatism, neuralgia, pain, nervousness, etc., are used to designate so great a variety of conditions that the patient should invariably be made to define what he means by them. Thus, as to "rheumatism," almost every painful affection of the entire body has been so designated by one patient or another ; "neuralgia," likewise, may mean any kind of pain—even that of rheumatism. The patient may say "pain" when he means a distress or a morbid sensation ; "nervousness" may be used to indicate general apprehensiveness, convulsions, tremor, sleeplessness, morbid fears, restlessness, and many other conditions.

The complete history of the case having been obtained, it is next in order to proceed with the physical examination of the patient. The methods commonly employed in the investigation of the conditions of different organs will be found in subsequent chapters, and need no additional mention at this time. The extent to which the physician should go in making these examinations will depend in great measure upon his judgment and experience. In most instances, examination of the organs which can possibly have a clinical relation to the subjective and objective symptoms will only be necessary. If, with such examination completed, all symptoms of the patient are fully accounted for, there is no necessity for proceeding further. But if, on the other hand, the diagnosis is incomplete, then it is necessary to make a systematic examination of all the viscera. Inasmuch as there are four clinical conditions or diseases capable of simulating almost any of the chronic diseases, namely, interstitial nephritis, hysteria, syphilis, and tuberculosis, it is important always to make examinations sufficiently complete, as a routine measure, to negative or confirm their existence. It may be that the condition for which the patient seeks relief does not come under one of the four mentioned, and yet one or more of these being present may greatly modify the clinical picture, and therefore the diagnosis or prognosis.

It should be every physician's duty to keep systematic records of all cases presenting themselves to him for treatment. It makes but little difference what system he employs, providing it is carried out. Such records are valuable in many ways. They cultivate proper methods of clinical work ; they enable the physician to refer to the past history of the patient, and to formulate his observations and present them as finished scientific essays to his *confrères*. If there is any one thing that is of paramount importance in making the finished clinician, it is the habit of keeping systematic and exhaustive records in all of his cases.

As to whether records shall be kept in books or according to the card system, there will always be differences of opinion. But it is the feeling of the writer, after seventeen years' experience with books and six years with the card system, that the latter is immeasurably the superior. Its advantages lie in the ease with which any record can be found ; in the fact that the entire record of a long case is in one place ; house-records may be kept with as much ease as those made in the office. The only valid objection thus far offered is that cards may be lost. But it is also possible for cases recorded in books not to be indexed, in which case the record is as good as lost. Temperature-charts and nurses' record-sheets may be made uniform in size and shape with the record-cards, adding greatly to the convenience of the system. If one has some favorite forms of record for particular cases, such forms may be utilized. He may have prepared for him charts for urinary, blood, and other examinations.

Opinions also vary as to the desirability of having printed examination-forms to be filled out. These are convenient, no doubt, but they waste valuable space, always an important matter to the busy man. Personally, I much prefer the plain sheet. To aid in making records, one may then follow set plans which he has prepared for himself.

In the Hahnemann Hospital, records are made on sheets devised by a committee consisting of Dr. C. M. Thomas and myself. On completion o the records, they, with the nursing-charts and other papers relating thereto, are filed in binding-cases. The record-sheets are arranged to be filled out with information concerning the following data:

Record No.........
Name...
Residence...
Married or single ....Age.....................................
Nativity ....Occupation...
Ward..............Bed...
Physician in charge...
Diagnosis...
Family history...
Personal history...
History of present illness...

Present Condition.

A. Subjective symptoms..
B. Physical examination. Date..
External surface ....
Head..
Trunk...
Extremities..
Circulatory system...
Gastro-enteric system..
Respiratory system..
Genito-urinary system...
Nervous system..
Special senses...
Special observations...
Pulse.........Temperature.........Respirations.........Weight...........

In filling out this record, the resident physicians are guided by the printed forms used in teaching the students record-taking under Dr. G. Morris Golden, and modified by Dr. F. Mortimer Lawrence from the schema presented by Hutchinson and Rainey. These forms read as follows:

HAHNEMANN MEDICAL COLLEGE AND HOSPITAL.

PRINCIPAL SUBJECTS OF INQUIRY IN MEDICAL CASES.

I. General Interrogation.

Name. Age. Occupation. Married, single, widow or widower. Address. Date.

1. Family History.

Parents.
Brothers and sisters.
Patient's own children.
} Ages. Health. Cause of death.

Diseases and causes of death in other relatives, especially from tuberculosis, gout or insanity.

2. Previous Health.

Nature and date of previous illnesses (especially gout, rheumatism or venereal diseases).

3. Environment.

Nature of work and its surroundings.

Home conditions, hygiene, etc.

Habits: tea, coffee, tobacco, alcohol; food, exercise, etc.

4. History of Present Illness.

How long ill? Supposed cause? First symptom? Sudden or gradual onset? Course of symptoms, in order of occurrence, up to time of examination.

II. Special Interrogation.

Following the above general interrogation, the patient should be questioned with particular care as to the one or more special physiological systems that may be diseased, as suggested by the facts already ascertained.

1. Alimentary System and Abdomen.

(a) If symptoms point to an affection of the stomach, inquire regarding—

Appetite.—Is it excessive, diminished or capricious? Does it increase on eating? Does he suffer with thirst?

Meals.—Their arrangement and the nature of the food. Does he eat between meals?

Sensations Referred to the Stomach.—Their nature, and exactly where felt. Are they produced or relieved by taking food? How long after eating do they come on? Are they specially influenced by different kinds of food?

Vomiting.—Frequency and time of; by day or by night; in the morning or in the evening? Its relation to eating; is it only after food, or does it occur at other times? Does it relieve pain or not? Does patient strain or retch much, or does the vomited matter come up quite easily?

GENERAL CHARACTER OF VOMITED MATTER.—Its amount and color. Is there ever "coffee-grounds" vomiting? Is it ever sour and frothy? Does it contain mucus or blood? If of food, how much has it been changed by the digestive processes?

ERUCTATIONS.—Presence or absence. Have they any taste?

FLATULENCE.—Presence or absence. Is it after food only, or between meals? Relation to particular articles of food? Does it tend to escape upwards or downwards? If upwards, its taste.

STATE OF BOWELS.—How often are they opened? Any special character of the motions?

(b) If symptoms point to an affection of the *intestines*, inquire regarding—

DIARRHŒA.—Its frequency, and its relation to meals or to special articles of food. Character of the stools. Has he ever passed any blood or slime? Is there any straining during defecation? Is there any flatulence?

CONSTIPATION.—What is his usual habit? Are the bowels opened regularly, and if so, how often? How long since the last motion? Has he ever noticed any grooving or flattening of the stools? Does the constipation alternate with diarrhœa? Has he any griping pain? Has he had any vomiting?

PAIN.—Character. Persistent or intermittent? Where is it felt worst? Is it aggravated or relieved by pressure?

(c) If symptoms point to an affection of the *liver*, *e.g.*, patient is jaundiced or has pain in the region of the liver, inquire regarding—

PAIN.—Its site. Has he ever had attacks of very severe pain coming on suddenly and lasting for a few hours? If so, did the pain radiate, and in what direction? Was there vomiting with it? Was he at all yellow after it subsided? Has he ever pain in the tip of the shoulder? Does he suffer with piles? Ever vomit blood? Has he noticed any change in color of urine or fæces? Does his skin itch at all (if jaundiced)? Inquire also as to his digestion, as outlined above.

2. Circulatory System.

If the symptoms point to an affection of the heart or blood-vessels, inquire regarding—

A family history of gout, rheumatism, angina, heart disease or apoplexy.

A *personal history* of rheumatic fever, chorea, scarlatina, diphtheria and other infections. (In children, inquire concerning sore throats and "growing pains.")

Subjective Sensations.—Dyspnœa. Has he to sit up in bed, or can he sleep lying down? Præcordial pain or distress: its exact site and character; does it radiate or not? If so, in what direction? Palpitation: its relation to meals and to exertion. Does the heart give an occasional thump? Sleep: good or bad. Does he dream? Giddiness: is it ever present, and when?

Signs of General Venous Distention.—Do the feet ever swell? Has he any cough? What is the state of the digestion? Does his nose ever bleed?

3. Blood.

If the symptoms point to an affection of the blood, inquire regarding—

Family history of bleeders. Has he had any loss of blood? Has he bleeding piles? What is the state of the bowels? If a woman —is menstruation excessive or diminished?

Subjective sensations of breathlessness on exertion; headache; giddiness. Do the feet ever swell?

Any possibility of lead-poisoning or malaria?

4. Respiratory System.

If the symptoms point to an affection of the respiratory organs, inquire regarding—

Family history of bronchitis, asthma, phthisis, or "scrofula." The patient's occupation: is he exposed to irritating fumes or dust? Has he ever had large glands in the neck? Does he sweat at night? Is he getting thinner?

Cough.—Its character and frequency; when is it worst? Does it pain him or not? Does he ever vomit with it?

Expectoration.—Its amount chaand general racter; yellow or not? Ever blood in it? If so, is it only after severe coughing? Is the blood bright and frothy, or dark in color?

Pain in Chest.—Is it aggravated by taking a breath? Constant or not? Where seated?

Dyspnœa.—When is it felt? If spasmodic, ask him to describe an attack.

5. Urinary System.

If the symptoms point to an affection of the kidneys or urinary passages, inquire regarding—

Family history of Bright's disease, apoplexy, or gout.

Personal history of scarlatina, syphilis, prolonged suppuration, lead-poisoning, gravel or gout, and previous renal disease.

Has he any pain in the lumbar region? Ever any attacks of acute pain shooting down into the groin?

Such remote symptoms as headache, drowsiness, vomiting, paralysis, convulsions, dimness of vision, dyspnœa.

Does the face ever look puffy in the morning?

URINE.—Is it altered in amount? Has he to arise in the night to pass it? Is it altered in color? Is it clear or turbid when passed? Ever any blood in it? If so, at what period of micturition is it present?

Is there any increased frequency of micturition? Is the increase by day or by night? Is there any pain during micturition? Is it before, during or after the act? What is its character, and where felt? Is it aggravated by movement?

6. Nervous System.

If the symptoms point to an affection of the nervous system, inquire regarding—

Family history of mental disease, chorea, convulsions, paralysis.

Personal history of rheumatism, gout, syphilis or alcoholism.

His work: is he exposed to any poisons, *e.g.*, lead, mercury, arsenic, etc.

In cerebral cases inquire as to any discharge from the ear.

Should he complain of convulsions, inquire as to—

Age at first fit? The supposed cause. Describe the first attack. When did the second occur? What has been the shortest and longest interval between the fits? Are they more or less frequent now? Do they occur during sleep? Has he any aura? Is the onset sudden or gradual? Does he remain rigid or does he struggle? Are the movements uni- or bi-lateral? Where do they begin? Does he bite his tongue, micturate or defecate during the fit? Is restraint necessary to prevent him from hurting himself? How does it end? Are there any after-symptoms, such as sleep, headache or automatism?

Should he complain of paralysis, inquire regarding symptoms of heart or kidney disease. (See Circulatory and Urinary Systems.) Had he any premonitory symptoms before the onset? Did it develop abruptly or gradually? Was he unconscious at the time; and if so, for how long? Has he any headache? If so, where is it situated? Is it worse at night? Has he any giddiness or difficulty in walking?

7. Bones and Joints.

If the symptoms point to an affection of the bones or joints, inquire regarding—

Family history of tubercular disease, rheumatism, gout or syphilis.

Personal history of tubercular disease, previous manifestations of gout or rheumatism, syphilis, gonorrhœa, any recent or remote injury; and in women, leucorrhœa or post-partum trouble.

If pain is referred to a bone, is it worse during the day or during the night? If pain is referred to a joint, is it constant, or only when the joint is moved? Are there any starting-pains at night? Is the pain affected by weather? Does the pain shift from one joint to another?

8. Children.

If the patient be a young child, it is necessary to question the mother as follows:

How many other children are there? Any dead, and of what? Where does patient come in the family? Have you ever had any miscarriages; and if so, when? How about the health of the father and yourself?

Was this a full-term child? Was the labor normal? Was the child breast-fed? If not, how fed? What food does it get now? Had it any rash after birth or any snuffles? When did it begin to get its teeth and walk?

III. Physical Examination.

Having interrogated the patient, proceed first to an investigation of the patient's general state, then to examination of the system most affected, and finally of each of the other special systems.

1. General Condition.

General state of consciousness, intelligence, development and nutrition. Decubitus, if in bed; or attitude and gait, if up. Expression of face. Pallor, cyanosis, jaundice, dropsy, trophic changes. Glandular enlargements. Character of respiration. Temperature.

2. Alimentary System.

Mouth.—Inspect teeth, gums, tongue, pharynx, fauces.

Stomach.—Palpate. Percuss. Examine contents (test meal or vomit).

Intestines.—Percuss. Palpate and inspect. Rectal examination, if necessary. Examine fæces.

Liver and Gall-Bladder.—Inspect, palpate and percuss.

Abdomen.—Inspect, palpate and percuss generally.

Spleen.—Palpate and percuss.

3. Circulatory System.

Præcordia.—Inspect. Palpate.

$$\text{Heart.—Percuss}\left\{\begin{array}{l}\text{upper border}\\ \text{right border}\\ \text{left border}\end{array}\right\}\begin{array}{l}\text{superficial.}\\ \text{deep.}\end{array}$$

Auscultate—
at apex.
tricuspid area.

- pulmonary area.
- aortic area.
- about third left interspace.
- veins and arteries of neck.

If a bruit is heard, note—

- Its time.
- Its character (harsh blowing, etc.).
- Its point of greatest intensity.
- Its direction of transmission.

PULSE.—Rate.

- Rhythm.
- State of vessel walls.
- Blood-pressure during and between beats.
- Sphygmographic tracings.

4. Blood.

Estimate hæmoglobin (Von Fleischl's hæmometer).
Count red and white corpuscles (Thoma-Zeiss apparatus).
Examine stained corpuscles microscopically.

5. Respiratory System.

Inspect chest (shape, expansion, etc.).
Palpate chest (expansion, vocal fremitus).
Percuss lungs (anterior, lateral, posterior).
Auscultate in same order. Note—

- Type of breath-sound.
- Character of vocal resonance.
- Presence or absence of accompanying sounds.

Examine sputum with naked eye.
with microscope (bacteria, connective tissue, etc.)

6. Urinary System.

Palpate kidneys.
Examine urine. Note—24 hours' quantity.

- color.
- specific gravity.
- reaction.

Chemical.

- amount of urea.
- albumin.
- sugar.
- phosphoric acid excretion.

Microscopical.

- crystals.
- casts.
- blood.
- pus.
- bacteria.

7. Nervous System.

Investigate (a.) Intellectual faculties (intelligence, memory, speech, sleep, delirium, coma, etc.).

(b.) Cranial nerve functions (tests).

FIG. 1.

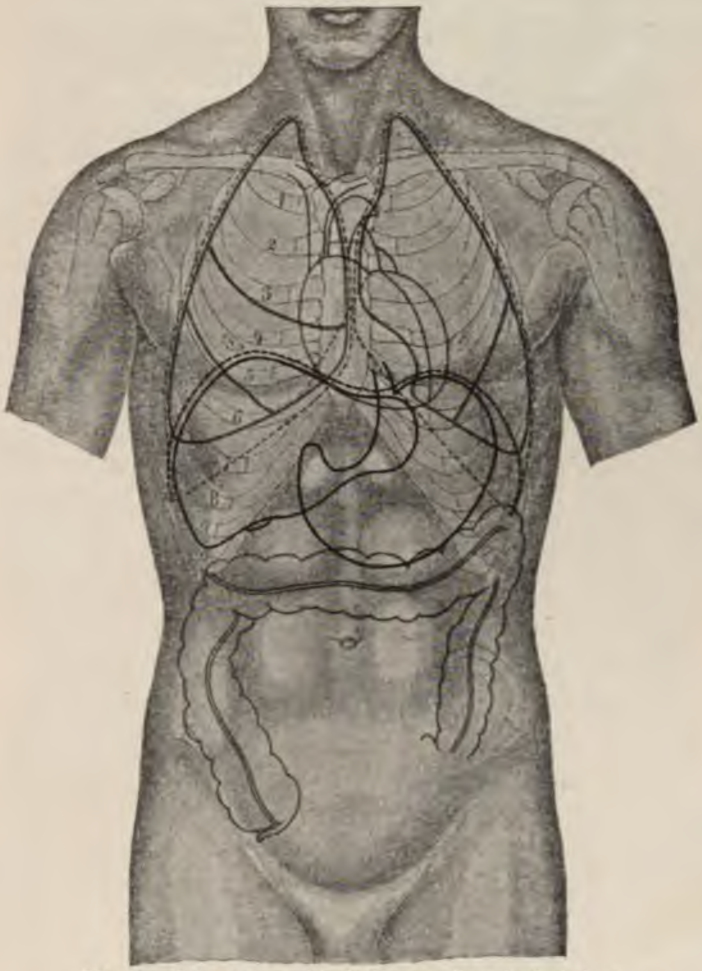

Diagram of front view of body for recording physical signs.

(c.) Motor functions.
- presence or absence of paralysis.
- abnormal muscular movements.
- state of muscular nutrition.

(d.) Sensory functions (touch, weight, temp., stereognostic and pain sense; abnormal sensations).

(e.) Reflexes.

(f.) Electrical reactions.

(g.) Fundus of eye (ophthalmoscope).

Of late, special diagrams have been introduced by Dr. Wm. C. Goodno, and are now in use for describing more specifically the various lesions. Such diagrams are of especial value in that they enforce greater accuracy in observation on the part of the examining physician.

Rubber stamps representing outlines of different portions of the body are on sale at the supply houses, and offer the same advantages as the printed diagrams. They possess the advantage of permitting any diagrammatic representation of the lesions present being placed on the same sheets as the balance of the record.

FIG. 2.

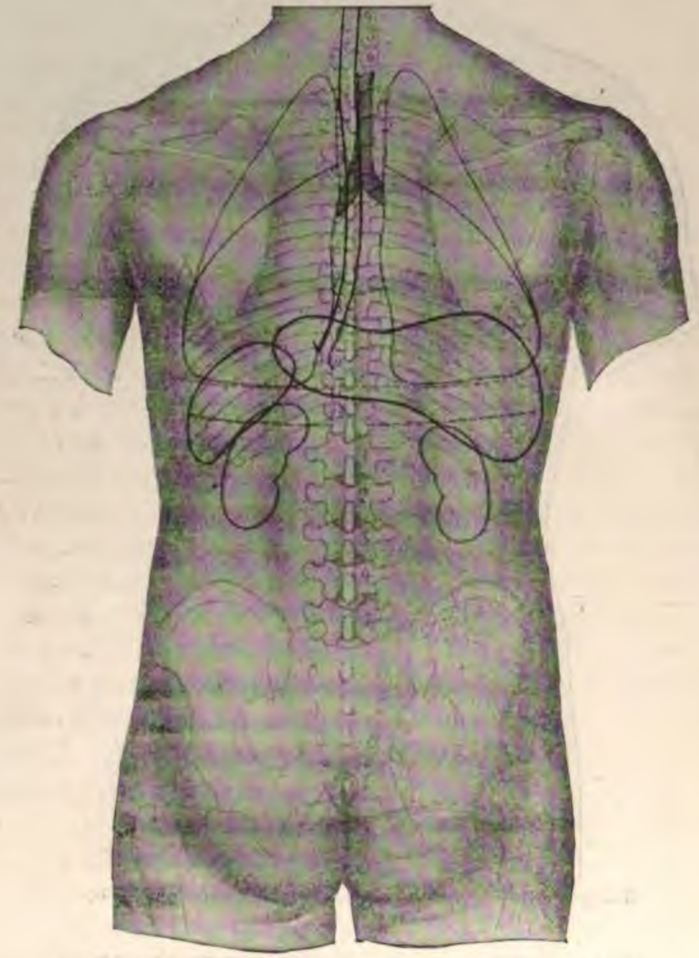

Diagram of posterior view of body for recording physical signs.

Equally important with the first record are the reports of the progress made by the patient from day to day. The information thus secured serves to confirm the diagnosis made at the first visit; or, if no definite conclusion is reached, it usually enables the physician to give a positive opinion. When conducting the subsequent examinations, it is wise to start the patient by asking some general question, such as, "How are you?" He will then give a fairly clear account of himself. If he does not cover all the ground concerning which information should be obtained, then the physician should ask specifically as to the condition of each of the symptoms noted on previous visits. If, at the first examination, the invalid is reserved, and the physician is unable to decide, for example, between gas-

FIG. 3.

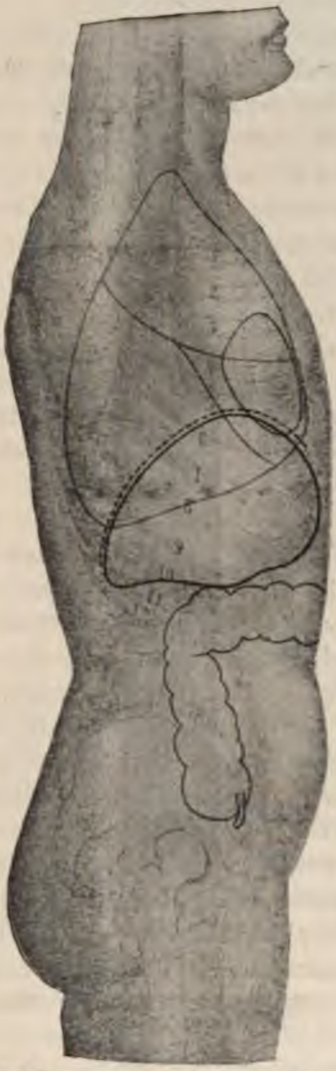

Diagram of right lateral view of the body for recording physical signs.

FIG. 4.

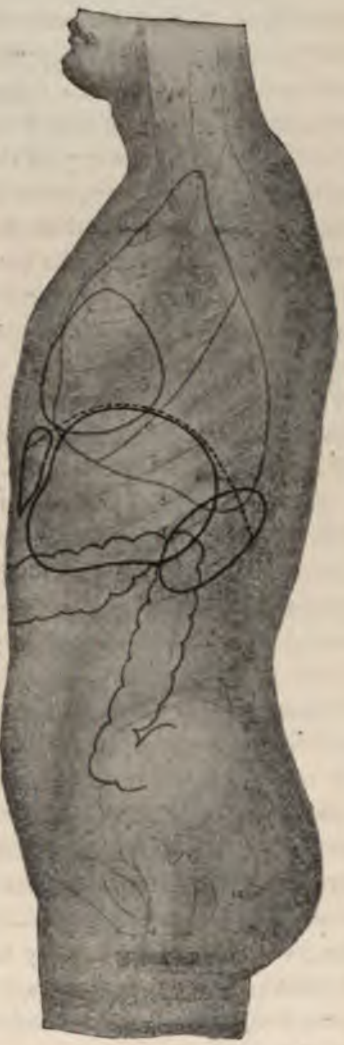

Diagram of left lateral view of body or recording physical signs.

tric ulcer and cancer, he should repeat his questions to establish their differentiation, in the hope that some positive information based on recent occurrences may be forthcoming. If the case happens to be one exhibiting physical signs which are capable of presenting marked variations within the interval of the consultations, the patient should be examined physically, as before. If, on the other hand, such variations are not possible, repeated examinations at short intervals are not wise, as they deprive the physician of the opportunity of noting the contrasts obtainable by examinations at longer periods. If the case happens to be one presenting a definite clinical course, as in the case of the acute infectious diseases, then inquiry should be directed to the discovery of the symptoms of the disorder as they appear from day to day. Thus, in scarlatina, the course of the rash, even to the termination of desquamation, the daily progress of the temperature, the appearance of the throat, the condition of the organs of special sense, but especially of the eyes, ears and nose, the presence or absence of albuminuria and lymphatic enlargements, the earliest manifestations of arthritic disturbances and endocarditis, should be systematically studied at each visit. It is a very wise practice, in making notes of the observations, to jot down negative findings. *Altogether too many otherwise excellent records are spoiled simply because the physician failed to record negative conditions.*

Take, as another illustration, typhoid fever. When attending a case of this disease, the physician should make it a routine practice to have four temperature- and pulse-observations taken daily ; he should investigate concerning the condition of the skin, especially as to the development of the characteristic typhoid exanthem ; he should note at once the presence of any œdematous swellings as indicative of venous thrombosis or nephritis ; the character of the muscular element of the heart-sounds should be a matter of daily study ; the state of the digestion and the quantity and character of food taken must be noted ; of paramount importance, because of the danger of intestinal hæmorrhage, is the character of the stools, the consistence and appearance of which must be noted on the nursing-chart after every movement ; the progress of the splenic enlargement should be a matter of daily note ; the condition of the liver should be studied from time to time, because of the liability to complications of this viscus in the course of typhoid ; the respiratory tract must be investigated as a matter of daily routine, because of the commonness with which typhoid is associated with bronchitis, and the liability to pulmonary hypostasis and œdema ; the frequency of nervous manifestations and their serious significance call for observation as to the mental condition, as it tends to stupor or delirium.

It may strike the reader that the making of examinations and records according to this complete system is a waste of valuable time. Experience will teach him otherwise. At first the work may go slow, but in a short time he will find himself able to make a reliable examination of the majority of cases inside of twenty minutes; and it is an exceptionally obscure illness in a very troublesome patient that will extend the period to forty-five minutes. The thoroughness of the investigation made on the first visit will greatly curtail the time required for subsequent consultation.

Records or day-books prepared by the patient are ofttimes of inestimable value. To illustrate: Not long ago I was consulted by a young woman whose main complaint was headache. A most painstaking examination failed to discover anything wrong in her ways of living, or any organic lesion. She was directed to send me a complete diet-list at the expiration of the week. Imagine my surprise to read such items as "ten potatoes," "eight slices of bread," as *parts* of certain meals. Twenty slices of bread represented the quantity of that article for single days. This same device has served me in equally good stead in numerous other cases, as it has others·to whom I have suggested it.

Before closing my remarks on the subject of record-making, I cannot refrain from saying a good word for shorthand as an aid to the physician. The foundation for the necessary dexterity can be laid in an incredibly short space of time; after which, practice will make perfect. Such records are illegible to prying patients; indeed, few but their owner can learn their secrets. The confidences of the consulting-room are thus made safer. To those who do not know shorthand, or will not take the time to master it, a system of private abbreviations will be found invaluable.

Concerning the Value of Certain General Information in the Diagnosis of Disease.

The Family History.—The family history may prove valuable by giving one an insight as to the vitality of our patient, as exhibited by the longevity and disease-tendency of his or her near relatives. It also enables us to formulate opinions as to the possibilities of special diseases which are inclined to run in families. In no class of cases is heredity more important than in the neuroses. In assigning inheritance as the cause in many instances, one must not jump to conclusions too quickly; for, after all, environment may be the principal etiological factor. Trying dispositions can readily wear out the constitutions of those with whom they come in daily contact. Our patients are too often constitutionally sound; their relatives need killing or curing.

Some diseases are transmitted indirectly—for example, tuberculosis. We all know this to be an infectious disease. We also know that some families are more prone than others to the infection. In other words, the constitution favorable to the propagation of tuberculosis is inherited. Of course, sight must not be lost of the fact that members of a family in which there is one case of this dread disease are constantly exposed to the danger of contracting the same by direct contact.

Arterio-Sclerosis.—The history of this condition in other members of the family will scarcely ever be obtainable from the patient as such. We may learn, instead, that members of the family age early in life, or that they suffered from hypertrophied heart, chronic Bright's disease, apoplexy, etc.

Hay-Fever.—Heredity is but a limited factor in the etiology of hay-fever. The principal cause of the disease is believed to be a hypersensitiveness to the pollen of ambrosia artemisiæfolia, this hyperæsthesia being the result of an inherited or acquired neurotic state. There is no doubt, also, concerning the increased liability of lithæmic individuals to this disease, and the tendency of lithæmia to run in families is well known.

Apoplexy.—As will be stated hereafter, the pathological conditions leading up to apoplectic seizures include arterial degeneration, hypertrophied heart, interstitial nephritis, and syphilis. The first three of these are inclined to run in families, although personal habits have considerable to do with their production. The occurrence of the hæmorrhage is purely a matter of accident.

Cancer.—There is a growing tendency to regard the transmission of cancer by heredity as rather problematical. Such assertions, however, are worthless, in view of the many instances observed by numerous physicians in which large numbers of the members of one family have been not only victims of cancer, but the malignant disease has actually invaded the same organ in each instance. Cases of this character must positively negative preconceived opinions to the contrary. A family history of cancer is obtainable in about 25 per cent. of the cases. The percentage is greater among women than among men. If both parents have suffered from the disease, the probability of affection of the offspring is thereby increased.

Friedreich's Ataxia.—This is regarded more as a family disease than one that is hereditary, for it occurs in several children of a family whose parents have not had the disease. The reason generally assigned for the non-hereditability of Friedreich's disease is that the victims seldom marry. Notwithstanding the remarkable tendency of Friedreich's ataxia to run in families, it must be remembered that isolated examples of it are observed from time to time. The first evidences of the disease are usually observed shortly after the child begins to walk.

Pseudo-hypertrophic paralysis, progressive muscular dystrophy and **spastic spinal paralysis** are examples of other affections attacking numerous members of a family. Spastic spinal paralysis of the hereditary type in particular gives a most remarkable example of the direct transmission of the disease, as shown by Bayley's cases, in which not less than five generations were known to have had the disease.

Diabetes.—The instances in which diabetes is hereditary are exceptional. Occasionally, however, we meet with cases giving a history of the disease in two or three preceding generations, while more than one brother or sister are likewise diabetics.

Epilepsy.—The transmission is rarely direct, probably owing to the comparative infrequency with which epileptics marry. In the majority of cases it will be found that there is a neurotic taint in the ancestors, as exhibited by a history of migraine, hysteria, neurasthenia, etc.

Heart disease is transmitted solely because of the hereditability of the etiological factors, as rheumatism, arterio-sclerosis, etc.

Hysteria, hypochondriasis and **insanity** are to be regarded as evidence of an unstable nervous organization, and as such are possessed of greater hereditability than any other conditions.

Syphilis.—Of hereditary syphilis as a disease-producing factor, it is my belief that the future will teach us much that is not now even dreamed of. When well-defined manifestations of the disease appear in early infancy, the diagnosis is clear; but when we meet with the so-called tardy cases, we truly have before us most difficult clinical problems. It is not an easy matter to obtain a history of the disease in a parent; for, however willing he may be to acknowledge his moral lapses when his own health is the subject of investigation, it is different when he is brought face to face with the enormity of the sin of bringing affliction to his innocent offspring.

When the knowledge of the present time concerning hereditary syphilis is compared with that of twenty years ago, and the many advances noted, it is easy to see how difficult it is to say what the future will bring forth. Certainly, we should not be willing to accept without reserve the statement of a prominent syphilographer, as follows: "Not only are the common tertiary maladies rare in the subjects of inherited syphilis, but the important class of what we may call aggressive degenerations, to which we attribute tabes dorsalis and general paralysis of the insane, are almost unknown in them." I know of no case in which tabes has been traced to parental infection, but already several undoubted instances of paresis have been reported as arising from the congenital disorder. The problem is a very complicated one, and not easy of solution. Many times I have observed cases of undoubted syphilitic disease in which everything favored the truth of the patient's denial of personal infection, and yet subsequent events confirmed the correctness of the diagnosis made.

Rheumatism.—The hereditability of rheumatism is universally conceded, and yet the present popular view that it is an infectious disease will probably do much to modify this opinion. The difficulties can be reconciled by assuming that there is a special diathesis favoring the incidence of rheumatism, and that this is transmitted from parent to child. It is also suggested that the poison upon which rheumatism depends is the special agency which is transmitted. Still others assert that it is some particular anatomical or structural peculiarity which is responsible.

Gout presents a most remarkable example of an hereditary disorder. Claims have been made for the transmission of the disease over a period of two hundred years. It is more than likely that such assertions have been made more for the purpose of establishing an aristocratic lineage than in the interests of medical science. Still the fact remains that the hereditability of gout is remarkable, and may extend over several generations. The luxurious habits of families of wealth account for many of these cases, but not for all. We know that there is a poor man's gout.

Age.—Age is an especially important factor in establishing a diagnosis. This may be well illustrated in cases of convulsions. This symptom, occurring in early infancy, is almost certainly dependent upon reflex irritation; occurring in childhood, it is probably epileptic; in young women, hysterical; in male adults between the ages of thirty and forty, syphilitic; at or beyond middle life, organic or renal. Of course this statement must be accepted as a generalization. Its value is suggestive, not absolute. However boldly we may assert certain diseases to be features of this or that portion of life, the fact remains that numerous exceptions are noted,—not sufficiently numerous, however, to impair the practical value of the general rule.

According to the time of life at which affections may assert themselves, the following arbitrary division may be adopted:

1. Congenital.
2. During infancy.
3. Childhood.
4. Puberty and adolescence.
5. Adults.
6. Middle age.
7. Advanced life.

1. The **Congenital** affections include those dependent upon the accidents of parturition and those arising from fœtal defects. Under the former heading may be mentioned atelectasis pulmonum, Erb's paralysis, and ophthalmia neonatorum. Under the latter, hæmophilia, hydrocephalus, idiocy and imbecility, malformations, nævi, progressive muscular atrophy, pseudo-hypertrophic paralysis, syphilis, and Thomsen's disease. The clinical manifestations of some of these may not appear until the patient is several years of age, *e.g.*, pseudo-hypertrophic paralysis and progressive muscular atrophy.

2. **Infancy.**—The affections liable to occur during infancy include the following general classes: Disorders of the stomach and bowels, respiratory catarrhs, skin affections, disorders of nutrition, reflex convulsions, and the acute infectious diseases. The principal ailment of the digestive system in infancy is diarrhœa, of which there are several varieties. (*Vide* chapter on Intestinal Symptoms.) They are especially liable to occur during the warm

months of the year, and are the most prolific source of infantile mortality. Of the respiratory affections, acute bronchitis and broncho-pneumonia are the most important. They constitute a serious source of infantile mortality during the winter months. Croupous pneumonia, though less common, may also occur. The skin affections common in infancy include eczema, intertrigo, seborrhœa, and strophulus. The disorders of nutrition are, for the most part, dependent upon dietetic errors. In some few instances, deliberate neglect is at the foundation of the trouble. More frequently, misdirected energy in abnormal directions is responsible. Infants, it must be remembered, may be starved by too much as by too little food. Bad results must follow the exhibition of food not adapted to the age for which it is prescribed. Methods of feeding also require consideration. The nutritive disorders of infancy include rickets, scurvy and marasmus (so-called).

The infectious diseases do not attack infants with the frequency observed in children ; probably because infants are not so much exposed to infection.

3. **Childhood.**—The general disease-tendency during childhood is in the direction of the infectious diseases, neurotic disturbances, tubercular affections, and disorders of the lymphatic glandular apparatus.

The infectious diseases to which children in particular are liable include scarlatina, diphtheria, measles, mumps, whooping-cough, roseola, rōtheln and varicella. In this list may also be included acute poliomyelitis anterior, which is now regarded as probably dependent upon infection. While children are undoubtedly more susceptible to infection than adults, the frequency with which they are attacked by these diseases is undoubtedly due in great part to their environment. The public schools afford unusual opportunity for the transmission of diseases. Moreover, children are proverbially democratic, and are not selective in their association with people or animals. Carelessness in the use of books, pencils, slates, etc., that have been in the presence of contagious cases, is undoubtedly responsible for the spread of contagious diseases.

The nervous system of childhood is proverbially unstable. This is exhibited by the frequency with which almost every febrile disorder is attended by restlessness, twitchings or delirium. This nervous instability is intensified by any unusual mental strain, as anxiety, fright, or excessive school work. Neurotic affections common in childhood include especially chorea and epilepsy.

Tuberculosis occurring in childhood attacks other structures than the lungs by preference. The lymphatic glands and the bones seem to be especially favored in this respect. Thus we find hip-joint disease and Pott's disease of common occurrence. As to the lymphatic swellings and their relation to tuberculosis, no one will question that such exist in many cases; but that the relation is invariable, as some would have us believe, is not to be entertained. Children certainly take on lymphatic enlargements much more readily than do adults.

Of the lymphatic affections, the one of especial importance is the enlargement of the post-nasal adenoid tissue,—a condition that is liable to produce serious disturbances in nutrition.

Rheumatism occurring in children exhibits a much milder type of joint-symptoms, as compared with the adult disease. At the same time, the liability to cardiac inflammations is much increased. Indeed, the latter may occur without any joint-involvement. The so-called "*growing pains*" are now recognized as an expression of acute rheumatism.

4. **Puberty and Adolescence.**—The nutritive changes taking place at this time of life favor the occurrence of nervous affections, especially in female subjects. Those of especial importance are chorea, epilepsy and hysteria. Syringomyelia,—a rare disease,—becomes manifest during the adolescent period. In boys, we note a general tendency to acne.

5. **Adult Life.**—Between the ages of twenty and forty, the general bodily nutrition is held to be at its best. We therefore regard this as the healthiest period, and during which disease should not occur excepting in response to some definite cause. With few exceptions, we observe diseases dependent upon disregard of general and personal hygiene and infections.

6. **Middle Age.**—With about the fortieth year the degenerative period of life begins, so far as the physical man is concerned. Mental development proceeds for many years thereafter, and should show no signs of retrograding until the threescore-and-ten period is approached. The various forms of malignant disease then become frequent, while the various affections dependent upon arterio-sclerosis constitute an important class of cases consulting the physician. Personal habits have, of course, done much to hasten the time at which the affections of middle age appear.

The affections especially common during middle life include angina pectoris, apoplexy, interstitial nephritis, cirrhosis of the liver, diabetes, the various forms of malignant disease, and paralysis agitans.

7. **Old Age.**—Vascular and respiratory affections are especially dangerous to the aged. This time of life is also liable to the same degenerative affections as make themselves more or less common during middle life. At this period we note as of especial frequency hard cataract, apoplexy, arterio-sclerosis, broncho-pneumonia, malignant disease, fatty degeneration of the heart, gout, paralysis agitans, prostatic disease, and thrombosis.

Sex.—Sexual predisposition to disease is largely dependent upon the different habits and environments of the sexes, though also, in part, upon the sensitively-organized or unstable nervous system of the female. Men, by reason of their occupations, are more liable to the ill-effects of exposure to the vicissitudes of weather and exertion; business cares bring worry and irregular habits of eating and sleeping. Their associations lead them to contract numerous habits. Women pursue an in-door or sedentary existence. Servants and domestic duties are the cause of considerable worry. Some live an aimless life, and this leads them to study their aches and pains. Indeed, with some women, this seems to be the principal pleasure of living.

The sensitive, nervous organizations of women exhibit their effects on all the complaints from which the sex suffers, and must be taken into consideration in the management of their ailments. The special nervous diseases to which women are liable are those included under the general terms hysteria and nervous prostration.

To be more specific in detailing the predominating tendency of each sex to diseases, the following lists are appended:

Males are more liable to vascular lesions, as aneurysm, arterio-sclerosis, aortitis, and hæmophilia; to cardiac diseases, as fatty heart, angina pectoris, hypertrophy, myocarditis, and valvular disease; to malignant disease in special locations, as the rectum, stomach and kidneys; to special nervous affections, as locomotor ataxia, bulbar paralysis, hypochondriasis, meningitis, general paralysis of the insane, pseudo-hypertrophic paralysis, syringomyelia, serratus magnus palsy, and spastic spinal paralysis; to the effects of bad habits, as alcoholism, tobacco and venery; to various disorders of digestion, especially to chronic gastritis; to renal lesions, as interstitial nephritis and cystic disease; to constitutional diseases, as gout, rheumatism and diabetes; and to liver diseases, as cirrhosis.

Women are more liable to the many neurotic symptoms included under the terms "hysteria" and "nerve prostration," as the acroparæsthesiæ, catalepsy, gastralgia, neuralgia, etc.; to floating kidney; to chorea, chlorosis, displacement of abdominal viscera, gall-stones, goitre, Graves's disease, mitral stenosis, myxœdema, and ulcer of the stomach; and to malignant disease of the breast and uterus.

Occupation exerts general effects as it requires its followers to lead a sedentary or active existence. An active existence, if exceeding physiological bounds, predisposes to vascular degeneration and rheumatism among the chronic diseases, and croupous pneumonia among the acute affections. A sedentary life leads to anæmia, constipation, liver disease, indigestion, gall-stones, gout, pulmonary tuberculosis, neurotic disturbances and gastric ulcer.

According to the special occupations, the following data may be noted (Leftwitch):

Bakers, grocers and *bricklayers:* Lichen agrius.

Painters, plumbers and *white-lead workers:* Lead-poisoning.

Cooks: Eczema, erythema and gastric ulcer.

Dusty trades, as *stone-cutters, miners,* etc.: Cirrhosis of the lungs, chronic laryngitis.

Domestic servants: Anæmia, gastric ulcer, erythema nodosum.
Skin-dressers and *wool-sorters:* Anthrax.
Chimney-sweeps: Epithelioma of scrotum.
Furriers: Arsenic poisoning, mercurialism.
Hatters: Mercurialism.
Looking-glass makers: Mercurialism.
Corn trades: Actinomycosis.
Bird fanciers: Psittacosis.
Divers: Caisson disease.
Clerks: Writer's cramp.
Elocutionists, public speakers: Laryngeal spasm, pharyngeal catarrh.
Telegraphers and *violinists:* Cramps.
Seamstresses and *shoemakers:* Tonic and clonic spasms.

Habits.—Aside from the conditions relating to occupation, and which have already received consideration, it should be our invariable rule to determine the habits of the patient as to clothing, eating, drinking, alcohol, tobacco and venery. Inquiry, however, but too frequently gives incorrect information, and yet the patient may regard himself as very truthful. This remark applies to all matters, and not particularly to those which the patient feels are a blemish to his character. I have seen children and adults kept in chronic ill-health simply because they were enveloped in entirely too many thicknesses of clothing. Too many people wear heavy flannels on days when they should be clad in the lightest of materials. But, as a rule, the patient not being aware of his shortcomings, the true state of affairs is to be determined only by actual observation on the part of the physician. On the other hand, there are persons, generally women, who go insufficiently clad during cold weather for no other reason than their objection to the clumsy shape imparted to their forms by heavy underclothing.

As to eating, it should be remembered that plain food, eaten properly at regular intervals, is a prerequisite to health. Too much or too highly-seasoned food is sure to bring about disorders of digestion or nutrition. Rapid eating throws too much work on the stomach, to say nothing of the irritation occasioned by the introduction of large, solid masses into that organ.

Drinking of water is hardly ever carried to excess, so far as the quantity taken is concerned. The usual difficulty with patients is that they do not take enough, or that they gulp down large quantities of ice-water during meals.

Alcohol is an important factor in the production of arterial degeneration, interstitial nephritis, cirrhosis of the liver, multiple neuritis, mental deterioration, chronic gastric catarrh, and delirium tremens. As a rule, the general appearance of the patient is such that it is easy to say, offhand, that he is given to the habit.

Excessive indulgence in tobacco produces catarrhal affections of the fauces and larynx, disordered digestion, irritable heart-action, general nervousness, and more or less cerebral exhaustion. All brands of tobacco are not equally active in these various respects. Those which contain the largest percentage of nicotine (domestic tobaccos contain as high as 15 per cent. as against 2 per cent. in the best Cuban) are the most deleterious. The effect of the weed in inhibiting mental activity does not seem to be generally recognized; but that it will have this effect is attested by the number of cases improving on cutting down their daily supply of cigars.

As regards excesses in venery, it is, as a rule, not so much the frequency with which people perform the act as it is the manner in which it is done. Altogether too many cases of nerve prostration are dependent upon faulty sexual hygiene.

History of Preceding Diseases.—The more extended one's experience in the clinical study of disease, the more he is impressed with the fact that each case presents, as a rule, a consistent totality of symptoms. Multiple conditions bearing no clinical relations to each other are exceptional. So it is when, studying a case, we nearly always find that what has gone before bears some relation to that which now exists. Certain diseases, which nearly every one has at one time or another in his career, disappear, and leave no ill-effects behind them in most instances. Such, for example, is the case with influenza. But there have been many cases of that disease in which the patient has subsequently suffered from myocarditis, nervous prostration, bronchial catarrh, etc. Scarlatina is sometimes followed by acute nephritis; diphtheria, by multiple neuritis; rheumatism, by endocarditis and valvular deformity; dysentery, by liver abscess; syphilis and tuberculosis are remarkable for the multiplicity of lesions which may follow in their wake. So important is the former as a disease-producing factor, and so frequent is it in all walks of life, that no case of chronic disease can be said to be complete without determining the patient's history as to the presence or absence of syphilitic infection. When dealing with women, information on this point can only be obtained by side questions as to eruptions, throat and mouth conditions, falling out of the hair and miscarriages, etc.

Environment.—The term "environment" is here used in the broadest possible sense, to include not only the place of residence, but the immediate home surroundings, the temperaments and general character of family and business associates, station in life, and numerous other points which affect the individual's state of mind. The influence of certain general localities in the production of special diseases is well known. Inasmuch as each locality has its own disease tendency, a specific tabulation of the affections common in each country would require too much space for presentation in a work like the present.

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The matter of immediate surroundings in their effect on the health of patients is, however, not sufficiently taken into account in investigating illnesses or in formulating plans of treatment. To illustrate: A few years ago there came to me a gentleman who occupied a responsible position, his employer being a man of unbounded energy and ambition, but of trying disposition. There could be no question concerning the effect of that temperament upon my patient. He had been generally regarded as a good example of the uric-acid diathesis. As soon, however, as he changed his employment his health improved, and he has remained well up to the present writing. Again, we meet with illnesses in persons who have been disappointed in their ideals; or in those whose ambitions or hopes have been shattered; or in those who are surrounded by nagging, disagreeable temperaments; or in those who have been shocked by the misdeeds of a dear and loved one. It is true that the knowledge of such a state of affairs is of comparatively little use in treatment, for we cannot prescribe a new household; but by such knowledge we do become acquainted with all the facts of the case, and we may prevent, what I have seen done many times in such cases, the performance of needless if not mutilating operations, as oöphorectomies, graduated tenotomies, sphincteric dilatations, etc. Moreover, a little sound advice, and the cultivation of a more philosophic spirit on the part of the patient, for it does not necessarily follow that but one party is to blame, will sometimes accomplish wonders.

CHAPTER II.

TEMPERATURE.

IN the systematic study of the various types of acute disease, it is universally conceded that especial attention must be paid to changes in the bodily temperature, pulse and respiration. A study of the first-named is important in that it, in many instances, furnishes important information leading to a correct diagnosis of the condition present, offers suggestions as to the condition of the patient from day to day or from hour to hour, and ofttimes indicates the onset of complications long before other clinical phenomena suggestive of such a mishap make themselves obtrusive. While the value of temperature-changes in the clinical study of acute diseases is generally admitted, they are practically neglected in the investigation of chronic diseases and in the study of so-called minor illnesses presented for treatment in the consulting-room. Thermometric observations under such circumstances are, in the vast majority of instances, unimportant when the illness happens to be of the nature suspected. But chronic illnesses are sometimes complicated by febrile manifestations pointing to the presence of some unsuspected pathological factor, and the minor ailments are often such only so far as subjective symptoms are concerned. The neglect of temperature-observations in either leads to the failure to recognize a serious malady (generally typhoid fever) early in its course.

As in all other matters, accurate technique is important in making temperature-observations. Neglect of proper precautions leads to a false sense of security or mistaken ideas. *The only true criterion of the state of the bodily heat is the clinical thermometer.* In years gone by, the sense of touch was commonly employed for the recognition of febrile conditions; and yet a more unreliable guide can hardly be imagined. The reasons for this are not hard to find. The perception of temperature-changes by touch is made by comparison, the standard of comparison being the physician's own hands. As these are cool or warm, so will he, by contrast, be likely to look upon the patient's surface as warm or cool. Thus it happens not infrequently that the patient's surface feels cool in the midst of high fever, and preternaturally hot when the actual temperature is but a degree or so above the normal.

Accuracy of observation, then, demands the routine use of the clinical thermometer. In order that this little instrument shall be used successfully, attention to the following points is absolutely necessary:

1. Accuracy of the instrument.
2. The allowance of sufficient time to obtain a correct reading.
3. Care lest the observation be taken in a part the seat of local disease.
4. Proper protection of the thermometer.

1. **Accuracy of the Thermometer.**—Attention to this point is not as important as it was in former years. Then the technical skill employed in the manufacture of instruments had not reached its present high point, and but few makers were regarded as reliable. It was then deemed necessary that all thermometers should be accompanied by a certificate of accuracy, the instrument of a well-known observatory being the standard. Now things are different. Competition has driven unreliable thermometers from the medical supply shops. The only possibility of inaccuracy remaining is found in the fact that thermometers change after a year or so, registering, then, about a degree above the correct temperature. This evil has been corrected in great measure by the manufacturers themselves, who properly season their instruments before graduating them.

2. **The Allowance of a Sufficient Time to Obtain a Correct Reading.**—The ordinary clinical thermometer requires to be kept *in situ* about five minutes before one can be absolutely certain that he has obtained a maximum reading. This point cannot be urged too strenuously. Of course, this long time is not pleasant for the patient. Of late years rapidly-acting thermometers have been placed on the market, it being claimed for them that a correct observation may be made in from thirty seconds to a minute. These instruments are certainly a great convenience. They are open to the objections of being more expensive, easily broken, and, when possessed of a corrugated bulb, more difficult of cleansing. When the convenience to the patient is considered, the extra care required to overcome these objections should not be allowed to interfere. Notwithstanding the claims of the makers, too much reliance must not be placed upon their rapidity. It is a good rule to allow about double the time stated.

3. **The Temperature Must Not be Taken at a Point the Seat of Local Disease.**—Errors arising from neglect of this point do not occur very frequently; still it is well to guard against them. It will never do to rely upon a temperature-observation taken in the mouth in cases of stomatitis; in the axilla when the axillary glands are inflamed; or in the rectum when there are inflamed hæmorrhoids or pelvic suppuration. Neglect of this precaution once caused me considerable anxiety. A case of typhoid fever was progressing favorably, when the temperature, for some unexplainable reason, began to rise. It was not until four days later that pain in the axilla suggested inspection of the part and the discovery of a small glandular abscess.

4. **The Thermometer Must be Properly Protected.**—Herein lies the most prolific source of errors in temperature-observation. The meas-

ures necessary to avoid these are better described when detailing the use of the thermometer in the various localities in which it is customary to make observations.

A few words in relation to thermometers with lens fronts may not be out of place. To some persons with deficient visual powers they are certainly a great convenience. If improperly constructed, as many of them are, the readings are made with great difficulty, unless the instrument be held in a particular position in relation to the eye and the source of light.

The places in which the thermometer may be placed for taking the temperature are:

1. The mouth.
2. The axilla.
3. The rectum.
4. The vagina.
5. The fold of the groin (in infants only).

1. **The mouth** has the great advantage of convenience. Persons of æsthetic tastes object to it, however, as they exhibit repugnance to this use of an instrument which has previously been they know not where. There is a growing tendency for families to possess their own thermometers,—a tendency which physicians will do well to encourage. If not thoroughly disinfected after use, infection may be carried from one case to another. This danger is to be avoided by immersing the thermometer in a 2 per cent. carbolic-acid solution and washing thoroughly. Correct observations in the mouth are only possible when the bulb of the thermometer is placed well beneath the tongue and completely covered by that organ, and when the stem of the instrument is well surrounded by the closed lips. In cases with marked nasal obstruction, this procedure may be impossible.

Temperature-observations are not to be taken in the mouth when patients are suffering with convulsions, delirium or coma.

Temperature-observations must not be taken in the mouth within half an hour after eating or drinking hot or cold substances. Accuracy really demands that mouth-observations should never be taken within half an hour after partaking of any kind of food.

2. **The axilla** is convenient for temperature-observations only in the case of patients confined to their beds. It is the most practical place in restless invalids and in children. The precautions necessary require strict attention. The axilla must be thoroughly dried. The bulb of the thermometer is next placed deeply in the axillary folds; the arm is then brought in close contact with the body, and somewhat over the chest. The muscles must be thoroughly relaxed; for if they are held rigid the soft parts are not likely to be in close contact with the thermometer. It would hardly seem necessary to caution the observer lest the bulb be inserted so deep in

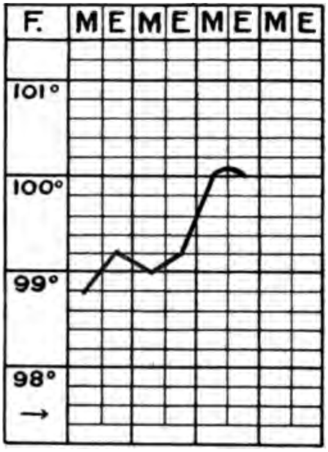
the axilla as to protrude posteriorly; and yet mistakes do arise from such self-evident carelessness.

3. **The rectum** is convenient only in comatose and convulsive patients, and is probably the only reliable place, when patients are suspected of malingering. Errors of observation may result from plunging the instrument into a fæcal mass. The bulb of the instrument should be inserted about two inches from the verge of the anus.

4. **The vagina** may also be selected in convulsive and comatose patients. It offers no advantages over the rectum.

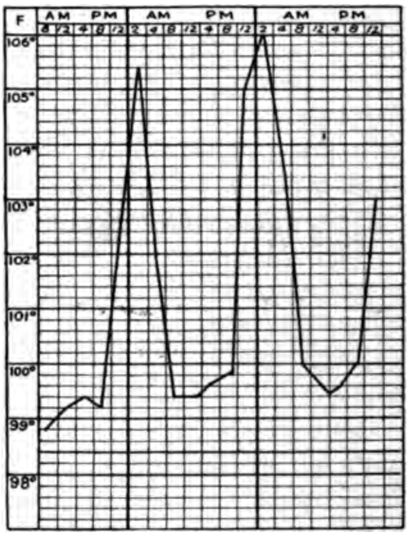
5. **The groin** is available in chubby infants. The bulb of the thermometer is placed well within the folds, and then the thigh is flexed firmly on the abdomen.

FIG. 5.



Temperature-curve of case of malignant endocarditis as it would have appeared from two temperature-observations daily.

FIG. 6.

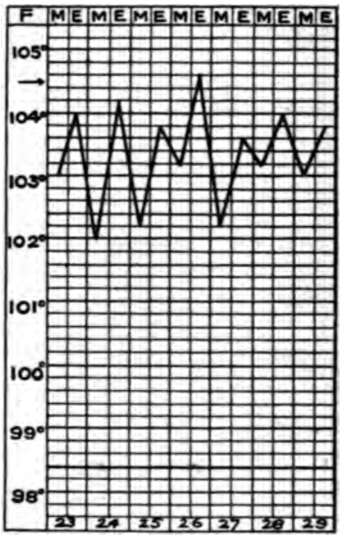


Temperature-curve of case of malignant endocarditis, six to seven observations daily. From same case as Fig. 5.

Temperatures taken in the mouth will usually be found to be about half a degree higher than those taken in the axilla, and those taken in the rectum and vagina half a degree higher than those of the mouth.

How frequently temperature-observations should be made is a question which every clinician will decide for himself. Prevailing custom teaches the propriety of two observations daily—one in the morning, and the other in the evening. All the popular temperature-charts make provision accordingly. More can be urged against the propriety of this plan than can be said in its favor. Circumstances which may influence the course of the fever are so numerous that the temperature not infrequently presents variations from hour to hour, sometimes when least expected. Compare, for example, the two charts herewith presented. (Figs. 5 and 6.) Both are given as records of the febrile changes in the same case,—one of malignant endocarditis. The preceding attendant had failed to recognize the presence of fever, his observations having been limited to the time of his visits, made usually in the early forenoon and early evening, at neither of which times did the temperature ever go higher than 99.5° F. At 1 A.M., however, there was always hyperpyrexia, the temperature reaching 105° to 106°. Another good illustration is shown in the annexed charts of the temperature-curve of a case of typhoid fever with phlebitis as a complication. Fig. 8 represents the febrile changes as indicated by the numerous observations taken. Fig. 7 is a chart based on records made at 8 A.M. and 8 P.M. Charts based on orthodox methods are valuable as giving a more general view of the course of the fever, avoiding the numerous departures from the general character of the curve by reason of various accidental influences. At the same time, they fail to take note of changes which may prove to be of the greatest clinical importance. Temperature-charts should, therefore, be based on not less than four records daily.

FIG. 7.



Temperature-curve of case of typhoid fever with phlebitis. Two observations daily.

There are two very practical objections to frequent temperature-observations. The first and most important is the danger of disturbing the patient's rest, which should be safeguarded under all circumstances, and the other is the mental effect of too much attention upon highly neurotic subjects. These are points which appeal to the individual judgment of the physician.

While the matter has no bearing on diagnosis, the writer cannot refrain from directing attention to a serious matter in relation to the standard temperature-charts in general use. With few exceptions, these are copyrighted by physicians. Their use in the sick-room serves to direct the attention of the family to their authors ; in other words, they are advertisements, and as such tend to disorganize the sick-room and its surroundings.

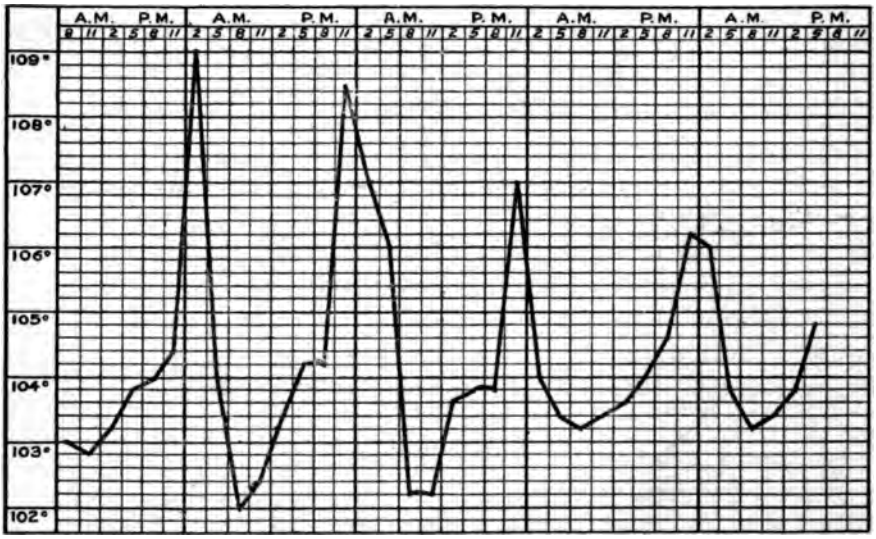
The normal bodily temperature is 98.4° F. Departures of one-half degree either way are not inconsistent with health.

This last-mentioned fact cannot be emphasized too strongly, as there are many well-meaning people of too anxious propensities who possess themselves of clinical thermometers, and, using them frequently, get families in a turmoil on the slightest departure from the accepted normal standard.

In this country, most thermometers are graduated in degrees Fahrenheit. Nevertheless, we find a growing tendency to use the Centigrade scale. Hence it is wise to be able to convert one into the other. To reduce degrees Fahrenheit to degrees Centigrade, subtract 32, multiply by 5, and divide by 9. To convert degrees Centigrade into degrees Fahrenheit, multiply by 9, divide by 5, and add 32.

Physiological departures from the normal standard may result from

FIG. 8.



Temperature-curve of case of typhoid fever with phlebitis; observations every three hours. Same case as Fig. 7.

1. *Taking of food.* There is not infrequently a rise of half a degree after taking a full meal, and this may continue during the digestive process.

2. *Exercise*, especially if violent, raises the temperature. If, however, the exertion is maintained for a long period, the reverse effect obtains, the patient becoming collapsic. Exercise of a severe nature cannot be regarded as physiological.

3. *Atmospheric heat.* In hot weather the temperature may be 99.5° F. Much depends, however, upon the degree of moisture of the air. Thus, in a dry atmosphere a very high temperature may be borne with comparative comfort, and without raising the bodily temperature, whereas a moderate heat with great humidity will soon bring about increased temperature.

4. *Age.* The temperature is slightly higher in infancy and lower in advanced life. The temperature of new-born infants ranges from 99° to

99.7°. In persons past middle life, it may be as low as 97° without being suggestive of illness. The temperature among children in general exhibits a higher degree of instability than among adults. Some children as a normal condition, according to Donald, show a tendency to maintain a constant high temperature, which in other children would be considered a pyrexia.

5. *Time of day.* The maximum temperature is attained in the afternoon or early evening; the minimum between midnight and early morning. In persons who work at night and sleep during the day, these variations are reversed.

6. *Mental excitement* may cause a rise of temperature to 100° F.

Fever.

Fever may be defined as preternatural heat. It is usually, though not necessarily, associated with quick pulse, thirst, loss of appetite, restlessness and prostration.

The febrile process has been characterized according to the intensity as

Slight when the temperature ranges between 99.5° and 101° F.

Moderate when from 101° to 103° F.

High when from 103° to 105.5° F.

Hyperpyrexia when above 105.5° F.

The Diagnostic Value of Fever.—The one certain diagnostic point concerning the presence of fever is that it indicates the existence of bad health. Of course, the illness must be one of those known to be capable of producing fever.

It is only exceptionally that the febrile changes, *i.e.*, the temperature-curve, is sufficient *per se* to establish a positive diagnosis. Not infrequently the temperature-curve enables us to say that the case in hand is an example of one of a limited class of diseases; nearly always we are enabled by its study to eliminate a very large number of diseases as having no possible bearing. In this, as in all other matters relating to diagnosis, we must remember that there is no such thing as a pathognomonic symptom, *i.e.*, a symptom having a single and invariable significance. Symptoms possess diagnostic and therapeutic value not because of themselves, but by reason of their associations. Even the absence of prominent or definite symptoms in association with fever must be regarded as of itself a symptom possessing a certain diagnostic value..

Certain acute infections are possessed in theory of a definite clinical course, the fever in these following a fixed and definite type. But the personal equation in the patient, aided by his surroundings, not infrequently brings about departures from the disease type. In this class of cases the character of the fever, with the associated symptomatic totality, establishes a diagnosis. This diagnosis having been made, the departures from the normal type of the disease become possessed of new diagnostic value,—*i.e.*, they have a positive significance which did not exist before the nature of the disease in hand was recognized.

The Prognostic Value of Fever.—In the abstract, it may be said that very high or very low temperatures are of serious significance. Much depends, however, upon the character of the disease present. If it be one in which high temperature is a part of the symptomatology, as in the febrile stage of malarial intermittent fever, then it cannot be regarded as an unfavorable indication. It shows simply that the patient is a very sick one. If the pyrexia, on the other hand, occurs in typhoid fever, it must be looked upon with increasing gravity as it rises in intensity. Much depends upon the duration of the febrile process. High temperatures of but an hour or so do not signify much ; those lasting for days are very serious.

Sudden changes in temperature must always be regarded as suspicious, if not actually dangerous. At the same time, it must be remembered that the significance of sudden changes depends in great measure upon the pathological condition present. A sudden fall in typhoid fever is ordinarily indicative of intestinal perforation or hæmorrhage ; a sudden fall in febrile affections disappearing by crisis is simply the natural favorable course of the disease. And so examples might be multiplied—all of which, however, are subject to exceptions.

A fresh febrile accession after the temperature has begun to fall generally indicates the advent of some complication.

The onset of fever in diseases not ordinarily febrile, *e.g.*, chorea, epilepsy, tetanus or cancer, suggests a complication or an approaching fatal issue.

It sometimes happens that the temperature in the course of fevers falls gradually without improvement of the associated symptoms. This process may continue until a fatal issue results. The fall of temperature in such cases is due to waning vital powers, with deficient thermogenesis. It can furthermore be observed that the surface-congestion and sweating so often characteristic of defervescence are wanting. There may, it is true, be a cold sweat present, but this is to be regarded as additional evidence of collapse.

The Nature of Fever.—The presence of fever necessitates some abnormalities in the functions which maintain the body at the temperature universally held to be the normal standard, *i.e.*, at 98.4° F. It is readily conceivable that these functions include

1. *The thermogenetic mechanism*, or that which governs generation of heat.
2. *The thermolytic mechanism*, or that governing the dissipation of heat.
3. *The thermotaxic mechanism*, or that which regulates the proper balance between the thermogenetic and the thermolytic mechanisms.

Heat-production is influenced in health mainly by muscular exercise and the quantity and character of food taken. Fats are especially active in this respect. Heat-production may be altered in response to the demands of the organism or to the requirements of heat-dissipation. Thus, large animals dissipate more heat than do smaller ones, and hence require a greater production, though this is not in proportion to their weight. Low external temperature makes a demand for greater production to maintain the normal standard.

Heat-dissipation is influenced by the covering of the body, the surrounding temperature, the amount of subcutaneous fat, the profuseness of sweat secretion, and the activity of respiration.

After all, it is heat-regulation, or thermotaxis, that is the most important factor in maintaining the normal temperature in health ; and it is disturbance of this mechanism that is the most important factor in the production of pyrexia. The controlling factor is in the nervous system, but the exact nature of this is not at all well understood. It is known that injury (experimental) to the following parts is capable of producing elevation of temperature : 1. Cortex : (*a*) The neighborhood of the cruciate fissure and just back of it (dogs and cats) ; (*b*) the junction of the supra-Sylvian with the post-Sylvian fissure (cats). 2. Basal ganglia : (*a*) Pons ; (*b*) corpus striatum (dogs, cats and rabbits) ; (*c*) optic thalamus (rabbits only).

The daily fluctuations of temperature in fever should now engage our attention. The same influences which bring about daily fluctuations in health are operative to a still greater degree in pathological states. With few exceptions, the maximum temperatures are observed in the after-part of the day, and the minimum in the early hours of the morning. On rare occasions we meet with cases in which the inverse type exists, for which, however, no adequate explanation has been offered. Excitement, dietetic errors and physical exertion cause rises in temperature to an extent hardly thought possible by those who have not had opportunities for clinical study. Children especially take on flights of temperature from very slight causes, probably because in them the thermotaxic mechanism is but poorly developed.

It is also well to remember that there is a large class of patients who exhibit high temperature in the presence of slight illness. The explanation of this lies in many instances in the previous condition of health of such subjects, though just as often it is so vague that we can use no better term than personal idiosyncrasy to account for the phenomenon.

Prevailing views contend that all fever is dependent upon some kind of toxæmia, although the acting poison is not always evident from a clinical standpoint. In some cases, as in the fever attendant upon simple fractures, such an underlying cause is far from evident. Clinically, we may say that fever attends upon

1. *Toxæmiæ and infections.*
2. *Inflammatory conditions.*
3. *Nervous lesions*, the clinical relations of which are not well understood. It is known, however, that lesions of the brain, *e.g.*, apoplexy, thrombosis, tumor, etc., are often associated with fever, even to the extent of hyperpyrexia.
4. *Peripheral nerve irritation* explains the marked pyrexia in certain painful affections with limited area of inflammation, the extent of the latter not being sufficient to account for the rise of temperature.

The Clinical Study of Fever.—Accuracy demands the analytical investigations of individual cases of fever. Each one must be studied according to the phenomena existing in individual stages, together with all associated phenomena. It is customary to describe fever as consisting of three stages, namely:

1. *The stage of onset*, or the invasion or initial stage.
2. The *fastigium*, or *acme*.
3. The *issue*, including defervescence, hyperpyrexia, collapse and death.

The initial stage demands a study of the mode of onset of the fever, whether gradual or rapid, the presence or absence of rigors, and the premonitory and associated symptoms.

The fastigium demands a study of its duration, the daily fluctuations of temperature, and the character of the associated symptoms.

Defervescence is the stage of decline of fever. It may take place by either *crisis* or *lysis*; by *crisis* when the subsidence of temperature is rapid, and by *lysis* when it is gradual. This is an important subject, because there are fevers in which defervescence by one or the other of these is characteristic. There are also fevers in which a sudden fall of temperature similar to that of defervescence by crisis is suggestive of a complication. The temperature-fall of crisis often amounts to as much as eight degrees in twelve hours, but it may take twenty-four or thirty-six hours. In any case it is practically uninterrupted. In defervescence by lysis the normal temperature is not attained until after the lapse of several days. The downward progress of the temperature is, as a rule, characterized by evening exacerbations and morning remissions. All cases in which the temperature falls are not to be regarded as pursuing a favorable course. Associated conditions must be borne in mind, for sometimes the temperature drops gradually, as in defervescence by crisis, the remaining symptoms of the disease showing no abatement; in fact, the patient soon dies. The fall of temperature in these instances is to be regarded as the result of failing vital powers.

An unfavorable termination of febrile cases is often characterized by more or less rapid rise of temperature to 105° F. or higher, such a condition being known as hyperpyrexia. Wunderlich has spoken of it as the "agonal" rise of temperature. Remarkable instances of this kind are within the experience of all physicians. Within twelve hours of death it is possible for the temperature to rise from 102° to 109° F.

While hyperpyrexia is always to be regarded as a serious matter, nevertheless its prognostic value must always rest with associated symptoms and conditions. Remarkably high temperatures have resulted in recovery. I myself have seen recovery ensue in a case of phlebitis complicating typhoid, the temperature reaching 109° F.; and Da Costa has reported a case of cerebral rheumatism with a temperature of 110° F., and Teale one of spinal injury with a temperature of 122 F., both of which ended favorably. Still, such cases must be regarded as exceptional.

Recrudescence.—This term is used to indicate a return of fever after the temperature has reached the normal point. In the majority of cases it is the result of some nervous excitement, over-exertion, or dietetic indiscretion. It may, however, be a forerunner of a true relapse, in which the disease runs its course over again *ab initio.*

Classification of Fevers According to their Temperature-Curves.—Fevers have been classified according to their temperature-curves as follows:

1. *Continued fevers,* in which the daily variations of temperature do not amount to much more than one degree.
2. *Remittent fevers,* in which the daily variations of temperature amount to two or more degrees, the temperature never falling to the normal.
3. *Intermittent fevers,* in which the temperature-course of the fever is interrupted by drops to the normal point.
4. *Relapsing fever,* characterized by an accession of fever lasting several days; then a period of apyrexia lasting several days more; then a return of the febrile stage, etc.

However desirable it might be to place each of the many varieties of fever under one of these headings, we are unable to do so with any positiveness because of the many departures from recognized clinical types. The most that we can do is to say that a given disease usually follows a certain type, but that it may also partake of one of the others.

The continued fevers include

a. Simple continued fever.
b. Typhoid fever.
c. Typhus fevers.
d. Pneumonic fever.
e. Erysipelas.

The remittent fevers,

a. Malaria.
b. Tuberculosis.

c. Puerperal fever, septicæmia and pyæmia.
d. Local suppurative processes.
e. Phlebitis.
f. The stage of decline of many continued fevers by lysis.

Intermittent fever occurs in

a. Malaria.
b. Typhoid fever.
c. Suppurative processes.
d. Tuberculosis.
e. Syphilis.
f. The anæmias.
g. Urinary sepsis, *e.g.*, cystitis accompanying enlarged prostate and catheter fever.
h. Hepatic intermittent fever.
i. Intermittent fever of morphia habitues.
j. Septic endocarditis.

The mode of onset of fever possesses great diagnostic value when one is assured of the correctness of the observations, for certain affections are characterized by sudden onset, while others appear gradually. It may happen, however, that a patient may ignore his illness for several days, so that when he gives his clinical history, it appears that his symptoms are of more rapid progress than is really the case.

Fever is of rapid or sudden onset in

a. Scarlatina.
b. Gastro-intestinal disturbance of children.
c. Pneumonic fever.
d. Malaria.
e. Inflammatory affections.
f. Typhus fever.
g. Influenza. (La grippe.)
h. Erysipelas.
i. Measles.
j. Variola.

Scarlatina and gastro-intestinal disease are practically the only affections capable of producing a sudden rise of temperature in children in the midst of good health without associated objective signs of local disease. The knowledge of the existence of an epidemic of scarlatina or of the exposure to the poison becomes an important differentiating factor. Scarlatina, moreover, is attended by a remarkable rise in the pulse-rate, *i.e.*, disproportionately rapid to the rise of temperature, high though the latter be. This is not likely to be the case in gastro-intestinal disorder. The case, in any event, cannot be long in doubt, for the characteristic eruption of scarlet fever appears on the day following the advent of the fever.

Symptoms Associated with Fever.—Increased bodily temperature is practically always associated with a definite series of symptoms, which include increased frequency of pulse and respiration, alterations in pulse-tension, and more or less disturbance of the secretory functions. There may also be emaciation or disturbances of nutrition and nervous derangements. These associated symptoms are, however, not equally prominent in all individuals with the same increase of temperature or in different pathological conditions attended by fever. One person, for example, may be able to pursue his ordinary avocations notwithstanding a temperature of 103° F., while another will be greatly prostrated with a temperature of 101° F. The personal equation is, therefore, an important matter.

The increased pulse-rate, as a rule, bears a definite relation to the height of the fever, each degree of fever corresponding to about eight to ten added pulse-beats. Notable exceptions to this are observed, *e.g.*, in typhoid fever, meningitis, scarlatina, diphtheria, and peritonitis. In the former two, the pulse-rate is disproportionately slow as compared with the temperature, while in the latter three it is disproportionately rapid. There are instances also in which the pulse rhythm is disturbed, irregularity or intermittence being observed. This phenomenon may be attributed to the effect of the toxæmia upon an already impaired heart, or of the effect of the toxæmia, *per se*, upon a neurotic subject.

The febrile alterations in arterial pressure vary with the stage of the disease. In most instances arterial tension is increased in the initial stage, owing, probably, to the increased frequency of heart-action without a corresponding relaxation of the peripheral vessels to accommodate the increased flow. In the fevers which continue for a number of days, arterial relaxation takes place, and the pulse becomes soft or of low tension.

The greater frequency of respiration is probably dependent upon the irritation of the increased bodily heat and the generated toxins upon the respiratory centres in the medulla, and in less measure upon the increased pulse-rate.

The secretory disturbances usually consist of diminished function, hence the skin and mucous membranes are preternaturally dry. Dryness of the mucous membrane of the mouth leads to thirst; that of the gastric mucous membrane brings about retarded digestion, and, in consequence, anorexia, and epigastric discomfort on taking food; that of the intestinal mucous membrane leads to constipation, with hard, dry fæces. The urine becomes concentrated; it is dark in color, and of high specific gravity. The necessity for careful dietetic precautions in fevers of all kinds is obvious.

Common concomitants of fever are also found in emaciation or wasting of tissue and disturbances of the nervous system. The former results not so much from the height of the temperature as from its long duration. Thus, emaciation is not prominent in croupous pneumonia, intermittent fever or scarlatina, in which diseases the temperature always runs high, while in typhoid fever, even when of a mild type, loss of flesh is a prominent feature. Akin to the emaciation is the degeneration of the heart-muscle and glandular tissues, which are of common occurrence in typhoid fever, and constitute one of the sources of anxiety during the days of convalescence.

The nervous disturbances of fever include sleeplessness and delirium. They are due to two sets of causes : one the effect of the increased temperature *per se*, and the other, the associated toxæmia. The personal equation already referred to is especially important in this connection, for there are patients who become delirious or semi-comatose upon the advent of the slightest rise of temperature, while others remain mentally clear throughout the long course of an exhausting and eventually fatal fever.

The relative importance of the pyrexia and the associated toxæmia in the production of the structural changes associated with febrile disorders is still a debatable question, though the trend of medical opinion is in the direction of the belief that these conditions are due almost exclusively to the latter. There is a growing tendency in the treatment of febrile disorders not to direct the principal efforts to the reduction of temperature. This change in procedure is the result of the increasing belief that pyrexia is a conservative process—a belief that modern bacteriological investigations have done much to foster. It has been demonstrated beyond all question that certain micro-organisms thrive better at the normal bodily temperature than they do at a temperature of 103° F., or more. As a matter of clinical observation, it is known that the most profound toxæmiæ accompanying the infectious diseases are encountered in the most severe cases,—the system not being able to react sufficiently to produce a fever. It is under exceptional circumstances only that there is real danger in the high degree of pyrexia ; instead, our chief concern should be the toxæmia.

Remarks on the Temperature-Changes and Clinical Course of the More Common Febrile Affections.

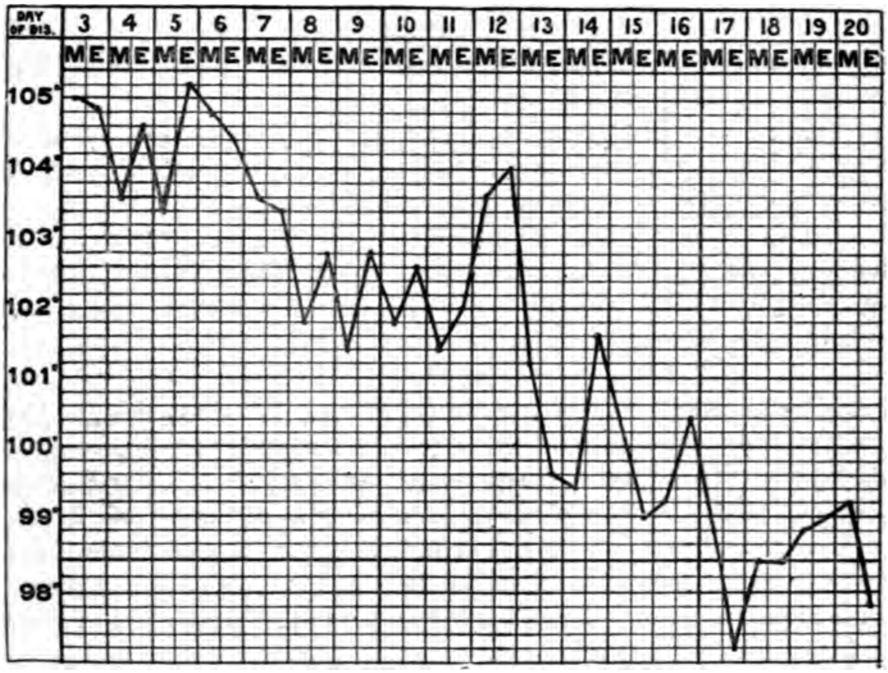
1. Simple Continued Fever.

The propriety of this term as designating an actual clinical entity is well open to question. For years it has been customary to apply it to cases of continued fever not associated with symptoms other than those of the febrile process *per se*, and apparently independently of any known specific infection or local pathological process. As skill in examination technique increases, such cases must become more and more rare. At present, this *so-called disease* is rarely diagnosed. Many of the cases claimed as such are probably examples of mild typhoids. Those of ephemeral existence probably arise from some auto-infection, *e.g.*, in the alimentary canal. Notwithstanding the above statement, I believe it is convenient to retain the term "continued fever" to designate a certain class of cases of non-specific origin, and apparently attributable in individual cases to heat, exposure, indigestion, fatigue, and nervous influences.

2. Typhus Fever.

Typhus fever is a highly-contagious disease occurring in epidemic form. The danger of infection increases in proportion to the closeness of contact with the patient and the defectiveness of the ventilation of the sick-room. Nurses and physicians in attendance upon the sick are very liable to be taken with the disease. The danger increases when there are many cases collected in one ward, while it is at a minimum when the patients are treated in tents, owing to the very free ventilation thus afforded.

FIG. 9.



Temperature-chart of case of typhus fever. (After Moore.)

The incubation period of typhus fever is about twelve days. In the diagnosis of the affection, the knowledge that an epidemic exists constitutes an important factor. The onset is sudden—even abrupt. First there appears a rigor or a succession of chills, and then the temperature rises rapidly to 103° or 104° F. the first day. It then remains as a continued type of fever, with morning remissions and evening exacerbations, until the seventh to the tenth day, when it disappears by lysis. The atypical course of the fever suggests asthenia or the presence of complications.

Associated with the fever are numerous symptoms dependent upon the profound toxæmia. These include headache, vomiting, general pains and aching, and great prostration. Delirium is frequently present, and may develop into a violent mania. Within three to five days after the onset of the fever the characteristic eruption appears. It is usually manifested at first upon the abdomen and upper part of the chest, later spreading to other portions of the body usually covered by the clothing. Two or three days are required for its full development. This eruption is commonly described as a "*mulberry-rash.*" It consists of a subcutaneous mottling, which soon becomes petechial, as evidenced by their failure to disappear upon finger-pressure.

With progress of the disease, the entire group of symptoms marking the typhoid state appear, including great prostration, rapid pulse, coma vigil, subsultus tendinum, dry and brown tongue, and failing heart. The urine is nearly always albuminous.

The most frequently-observed complications include broncho-pneumonia, terminating in some cases in gangrene of the lungs, noma, pyæmic abscesses, gangrene of the extremities, and hæmatemesis.

Typhus fever is to be regarded always as a dangerous disease. The mortality varies with the epidemic, sometimes amounting to as high as 50 per cent. The usual time for the fatal issue is towards the end of the second week.

3. Typhoid Fever.

The characteristic temperature-curve of typhoid fever as described by Wunderlich rises by steps, with morning remissions and evening exacerbations, during the first week. The fever is then supposed to have reached its height. The fastigium proceeds for another week, during which time the fever is practically continuous, or with slight evening elevation. The beginning of the third week marks the onset of the stage of decline, which, like the initial stage, is gradual, the temperature becoming normal at about the twenty-first day of the disease.

Exaggerated diagnostic importance has been attached to the Wunderlich curve, especially in the past, when it was not uncommon for some physicians to insist upon its presence before a diagnosis of typhoid fever could be established. It may be that the typical uncomplicated typhoid fever always follows this course; but when one thinks of the many surrounding conditions which tend to influence temperature, and the modifying effects of medicinal and hygienic measures, he cannot expect many cases to follow what has been alleged to be the orthodox type.

In the majority of cases, perhaps in all, the fever is of gradual onset, not attaining its maximum until the seventh day or thereabouts. Many cases are observed which are apparently not in accord with this statement. In some, it may be due to gross negligence on the part of patients and friends; in the majority, however, the suddenness of the rise of temperature is apparent rather than real, because the fever is not recognized until it has existed for a number of days.

Morning remissions and evening exacerbations are present in practically all cases. Exceptions are observed in what has been called the inverse type of typhoid fever, in which the exacerbations are in the morning. It is a matter of general experience that such cases are rather more serious than ordinary.

As a rule, the fluctuations in temperature from one part of the day to another do not amount to more than two degrees; and yet cases in which the temperature-range is over four degrees are not very uncommon. Theoretically, these remarkable remissions should lessen the deleterious effect of persistent high temperature upon the tissues at large. Nevertheless, it is important to know that they have been observed in cases having a serious outcome.

We expect the fever of typhoid to disappear by lysis. In exceptional instances, which have not been fully explained, the temperature has fallen rapidly to the normal and convalescence has been established. Take, for example, one of my own cases. The patient, a man aged 42 years, had a sudden fall of temperature amounting to four degrees, followed within a few hours by the appearance of bloody stools. He rallied nicely, and the disease was pursuing a normal course for such a severe case until, one week later, the temperature fell from 103° to 97.5° F. inside of four hours without a single unfavorable symptom. Fresh hæmorrhage or perforation was suspected, but no new symptoms developed. There was not even another accession of fever, and convalescence was uninterrupted. Rosenthal reports a similar case, in which, on the fourteenth day, the temperature dropped to 95° F., remaining subnormal for four days, after which the disease pursued a characteristic course. There was no evidence of hæmorrhage or perforation.

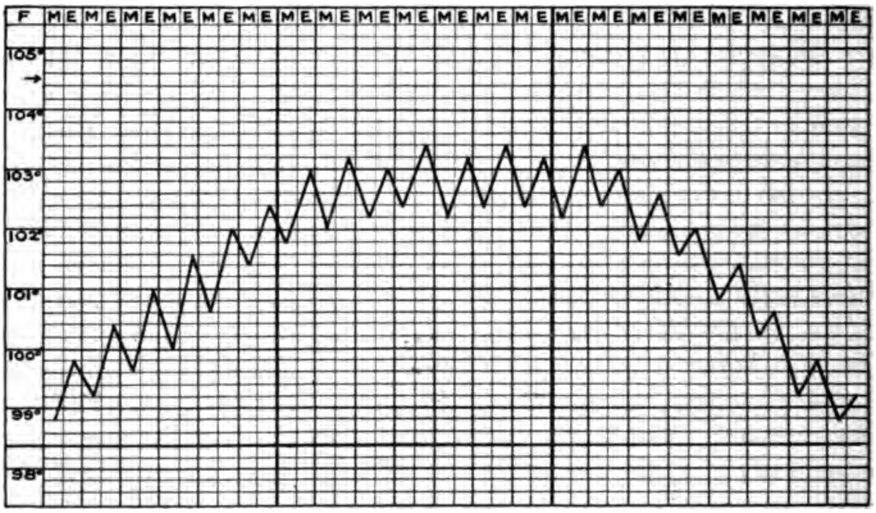
Very remarkable are those cases of typhoid fever which run their course without rise of temperature. It is true that such instances are rarely observed, but that they do occur is attested by excellent clinical authority and post-mortem examinations. The general consensus of opinion favors the idea that these cases offer a less favorable prognosis than do those running a typical course.

Accepted teachings concerning the defervescence of typhoid fever provide for a gradual return of the temperature to the normal standard. This is correct for the vast majority of cases. Exceptionally, however, we observe instances in which the fever disappears rapidly, as in the case already described. While, ordinarily, defervescence is complete by the end of a week after its commencement, cases are not infrequently observed in which a tantalizing slight pyrexia persists for a number of days beyond that period; indeed, it would seem to the medical attendant that the fever will never disappear. Such extensions of the fever are probably not all explainable on the same hypothesis. In some instances they are due to post-typhoid lesions, but in many more to the starvation-diet to which the patient has been subjected for weeks; at least this fever very often disappears as soon as the patient is allowed to take some solid food.

Sometimes the defervesence of typhoid fever is characterized by an intermittent fever, the temperature being normal in the morning and two or three degrees above the normal in the evening. No satisfactory explanation of this phenomenon has been offered. Certainly malaria is very rarely at its foundation.

However frequent the so-called typhoid-fever curve of Wunderlich may be, departures from it are sufficiently common to lead us to reject it as of much diagnostic value. Wunderlich's general rule is quoted approvingly by Dreshfeld: "Any fever which on the second day reaches to 104° F. is not enteric fever, nor is it enteric if the fever does approach 104° F. on the evening of the fourth day; on the other hand, enteric fever may be diagnosed if, in a middle-aged person suffering from an acute febrile attack, the evening temperature on the fifth day or within the first week is between 103° and 105° F., and alternates with morning temperatures which are 1.4° to 1.7° lower, unless some other disorder can be discovered to explain the height of the fever."

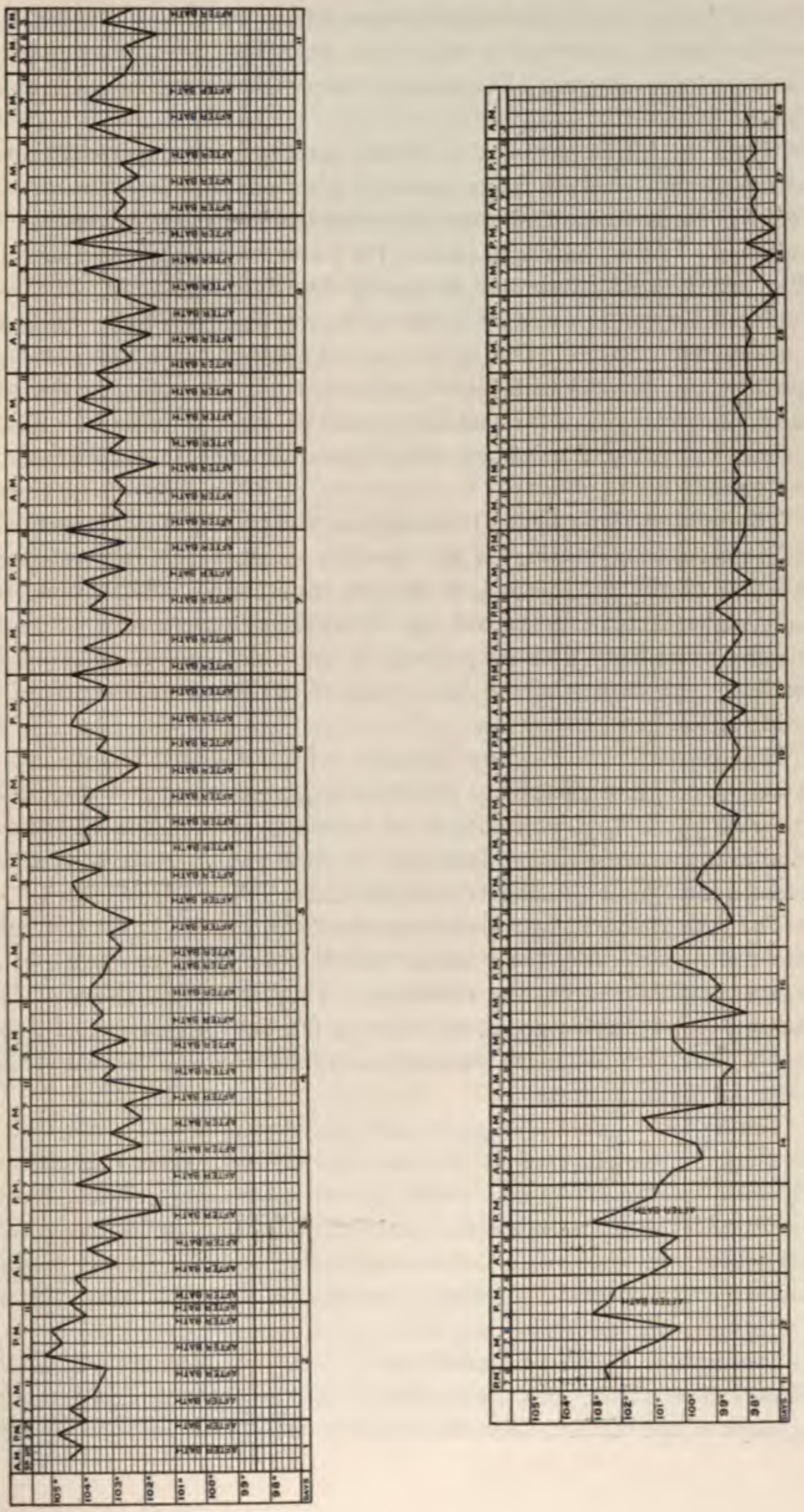
FIG. 10.



The temperature-curve of typhoid fever according to Wunderlich's teachings.

Instead, we may say that any fever continuing for a week without sign of abatement, no matter what its course may have been, will sub-

FIG. 11.



Temperature-chart of an unusually severe case of typhoid fever, treated in the Hahnemann Hospital, terminating in recovery.

sequently be proven to be typhoid,—providing, of course, there are no physical signs or symptoms to show it to be otherwise. In other words, the vast majority of cases of continued symptomless fever are examples of typhoid.

While the febrile phenomena attract great attention, typhoid fever is not without numerous other symptoms of great practical importance. One set of symptoms demanding constant attention are those relating to the circulation. In the prodromal period, the pulse may exhibit an undue irritability, as shown by increased frequency on slight exertion. The usual condition, however, is increase in the pulse-rate, but not in proportion to the rise of temperature. During the second week of the disease, the pulse frequently runs parallel with the temperature-curve, excepting in the presence of some complication or undue general or cardiac asthenia. Almost from the beginning, the intravascular tension is low, as exhibited by the dicrotic pulse.

Throughout the course of the disease the condition of the heart demands careful study, owing to the possible onset of parenchymatous degeneration of the myocardium or of true myocarditis. These conditions usually make themselves known by the ordinary phenomena of cardiac weakness, as rapidity and irregularity of the pulse, enfeeblement of the apex beat, and dulling of the first sound of the heart. Dilatation of the left side of the heart is present.

Enlargement of the spleen becomes evident towards the end of the first week, and proceeds rapidly until that organ attains two or three times its normal size. This symptom is so regularly present that it is now regarded as a necessary clinical feature of typhoid fever. As a rule, it does not lessen until the beginning of convalescence.

The rash makes its appearance on about the seventh to the tenth day. It consists of so-called "rose-spots," which have been compared, in appearance, to flea-bites on the abdomen. They are rarely absent. They come out in successive crops, and after a few days disappear. In some cases they are very abundant, and may cover not only the entire abdomen, but the chest and extremities.

The condition of the urine in typhoid fever does not seem to have been studied with the care its importance merits. It is now known positively that infection may be carried by this excretion. Albuminuria is the rule, rather than the exception. It is usually dependent upon the fever. It may, however, be the result of a complicating nephritis (*nephro-typhoid*). Pyuria is not infrequent. It usually persists for some time after recovery, but ultimately disappears.

The specific lesion of typhoid fever being an ulceration of Peyer's patches, lesions of the gastro-intestinal tract are to be expected. In the early days of the disease, these include only such disturbance of digestion as one would expect to accompany a febrile disorder, as loss of appetite, coated tongue, some abdominal discomfort, and constipation. But in the fully-developed disorder, other phenomena, as tympanites, diarrhœa and intestinal hæmorrhage, appear. The tympanites occurs for the most part in severe cases, especially in those which have not been taken in hand early in the course of the disease. It is to be regarded more as evidence of the profoundness of the intoxication, rather than of the extent of the ulceration.

While gurgling and splashing sounds, on palpation over the cæcal region, are commonly found in cases of typhoid fever, they must never be accepted as important diagnostic symptoms.

Ehrlich's diazo-reaction appears after the first week of the disease. When first described, it was claimed for it that it was an important diagnostic symptom. But it is found in numerous other conditions, and the lateness of its appearance in the course of typhoid makes its diagnostic value very problematical. (*Vide* chapter on Urinary Symptoms.)

Cases of typhoid fever may run their entire course without diarrhœa. In the majority of cases, however, we expect that symptom to be present. Attempts have been made to describe a characteristic typhoid stool. In reality, the only uniform characteristic is the presence of the typhoid bacilli. The macroscopic appearances of the bowel movements exhibit the widest possible features. In many instances they present the appearances of ordinary catarrhal diarrhœa. In many others they may aptly be described as like "pea-soup." The frequency of the loose movements is not usually excessive. Four to six stools daily probably represent the average, while ten or twelve stools daily are discharged only in severe cases.

Intestinal hæmorrhage takes place in about one-sixth of the cases of typhoid fever. This accident may result from one or two lesions. Occurring early in the course of the disease, *i.e.*, during the second week, it is the result of oozing from the swollen and friable tissue of Peyer's patches. When, however, it appears during the third week of the disease, it is dependent upon ulceration and exfoliation of sloughs. In either case, the amount of blood lost may be quite large. The appearances of the stools in cases of this accident present great variations, according to the quantity of blood lost and the length of time it is retained in the intestinal tract before its evacuation. There may be but a slight reddish tinge imparted to the stools; or, because of the long retention of the blood, it may undergo changes, and appear in evacuations as black, tarry masses. The onset of hæmorrhage is usually characterized by a sudden fall of temperature. The occurrence of hæmorrhage does not bear any relation to the severity of the attack, as most serious losses of blood have occurred in cases which have excited no apprehension up to the time of the discharge of the bloody stool. The alarming character of the symptom usually leads to exaggeration of description on the part of attendants. It is the rule for but two or three bloody evacuations to take place, and these following closely upon one another. Very exceptionally, eight or ten bloody stools may be discharged. Cases presenting this complication are always to be regarded with some concern, about one-fourth of them terminating fatally. The causes leading up to intestinal hæmorrhage are usually found in carelessness in the early days of the disease, dietetic errors, and restlessness on the part of the patient.

The symptoms attendant upon intestinal hæmorrhage are generally those associated with loss of blood from other portions of the body. Sometimes there is attendant abdominal pain.

A far more serious complication of typhoid fever is *intestinal perforation.* The advent of this accident is likewise marked by a sudden fall of temperature, with symptoms of collapse, followed by those of peritonitis, which may be localized or diffuse. The majority of cases end fatally. Sudden and severe abdominal pain is the rule. The determining causes usually assigned for perforation include improper feeding, undue exertion, vomiting, and excessive tympanites.

Hepatic complications are occasionally observed. These include jaundice, abscess of the liver, and inflammatory affections of the gall-bladder and bile-ducts. The jaundice may be catarrhal in origin by reason of the extension of the catarrhal inflammation of the duodenum to the gall-duct, toxic, or symptomatic of abscess or disease of the gall-bladder. The majority of cases of mild jaundice attendant upon typhoid fever probably belong to the first-named variety.

Among the respiratory symptoms attendant upon typhoid fever, bronchitis is observed with such frequency as to be placed among the regular phenomena of the disease. In some cases it is very severe, and may constitute a matter of considerable concern to the medical attendant. The subsequent development of pneumonia of either the croupous or catarrhal variety is to be regarded as a true complication, but is, fortunately, rare.

Very few cases of typhoid fever run their course without numerous symptoms referable to the nervous system. In the early days of the disease, headache is almost invariably present. It bears no relation to the severity of the attack. The pain is usually of a dull and heavy character. There may also be co-existent backache, soreness of the muscles generally, and great restlessness. Later, there appears mental hebetude with dullness of the senses. This sometimes takes the form of somnolence. Towards the end of the first week or beginning of the second, delirium appears in probably half of the cases. This is usually worse at night, and is characterized by rambling, incoherent speech, low mutterings, or, in extreme cases, by actual mania, with violence of speech and action. In many cases there is picking at the bedclothes, grasping at the air, subsultus tendinum, etc. Tremor is not unusual, and is commonly an expression of the asthenia present, though it is regarded by some as evidence of the extent of the intestinal ulceration. It is especially liable to occur in young subjects, and in those who have been addicted to the use of alcohol. The parts affected are generally the face, lips and tongue.

In exceptional instances the delirium is of such sudden onset and of so violent a character as to lead to the suspicion of the case in hand being other than typhoid fever.

Ordinarily, the diagnosis of typhoid fever should not be attended with any difficulty. The principal errors arise in the ambulant and abortive cases. Other diseases, notably appendicitis, pneumonia and meningitis, may so simulate it as to make the diagnosis a matter of the greatest difficulty. Trouble also arises in cases of typhoid in which some one set of symptoms, as those of the respiratory tract or of the gastro-enteric system, predominate over all others and obscure the original disorder. In all of these cases we have in the Widal reaction a most reliable sign. (*Vide* chapter on The Blood.) This test will certainly enable us to recognize such anomalous forms of the disease as those without fever and those occurring independently of intestinal ulceration.

The possible complications and sequelæ of typhoid fever are many, though they are relatively infrequent in cases which have been treated properly from the very beginning. They include multiple neuritis, myocarditis, endocarditis, ocular paralyses, neurasthenia and hysteria, psychoses, pleurisy, tuberculosis, abscess of the lungs, pulmonary gangrene, lobar pneumonia, suppurative cholecystitis, periostitis, pyelo-nephritis, chronic nephritis, and many others.

In formulating a prognosis during the course of typhoid fever two sets of symptoms are of especial value. They are those relating to the temperature and the pulse. In no disease is persistent high temperature of more serious significance than in the one under consideration. And yet one should not be led into formulating a prognosis on the temperature alone. A maintenance of the fever above 103° F. for days at a time, without marked remissions, should be regarded with some concern. If this fever should not be associated with nervous symptoms or rapid pulse, it need not be regarded with any alarm. One of my patients ran a temperature between 105° and 106° F. for several days, and made an uneventful recovery; but at no time in the course of her illness did she have a serious symptom other than the high fever. The supervention of nervous symptoms, as delirium, etc., always adds to the gravity of the situation. When the delirium persists through the day, and is attended by jactitation of muscles, dry and brown tongue, the outlook is unfavorable. The condition of the heart is of the utmost importance. As long as the pulse-rate keeps within 120 per minute at the height of the disease, one may feel safe so far as the condition of the heart is concerned. Weakening of the muscular element of the heart-sounds calls for strict attention to that organ. The gradual moistening of a dry, brown tongue is always a hopeful sign.

4. Pneumonic Fever; Croupous Pneumonia.

Uncomplicated croupous pneumonia is characterized at its onset by a more or less pronounced rigor, with rapid rise of temperature to 104° F., or higher. This fever begins with the very inception of the disorder. It is quite apt to be associated with severe headache and pain in the side. The fever continues, subject to the usual diurnal variations, until the seventh to the ninth day, when it disappears by crisis. It not infrequently happens that the temperature becomes subnormal during convalescence. The crisis of pneumonic fever is sometimes preceded by a pseudo-crisis some twenty-four to forty-eight hours.

Numerous departures from the above-described type occur, the most remarkable of which are the afebrile cases, and in which the temperature remains at practically the normal point throughout the entire course of the illness. Such cases are apt to pursue a tedious course, and are to be diagnosed only by the physical signs. They occur principally in the aged, in the victims of chronic Bright's disease, and in those who have long been addicted to alcoholic excesses.

Pneumonic fever does not always disappear by crisis. Cases are not uncommon in which the temperature falls gradually. The majority of these present typhoid symptoms and pass through a tedious convalescence.

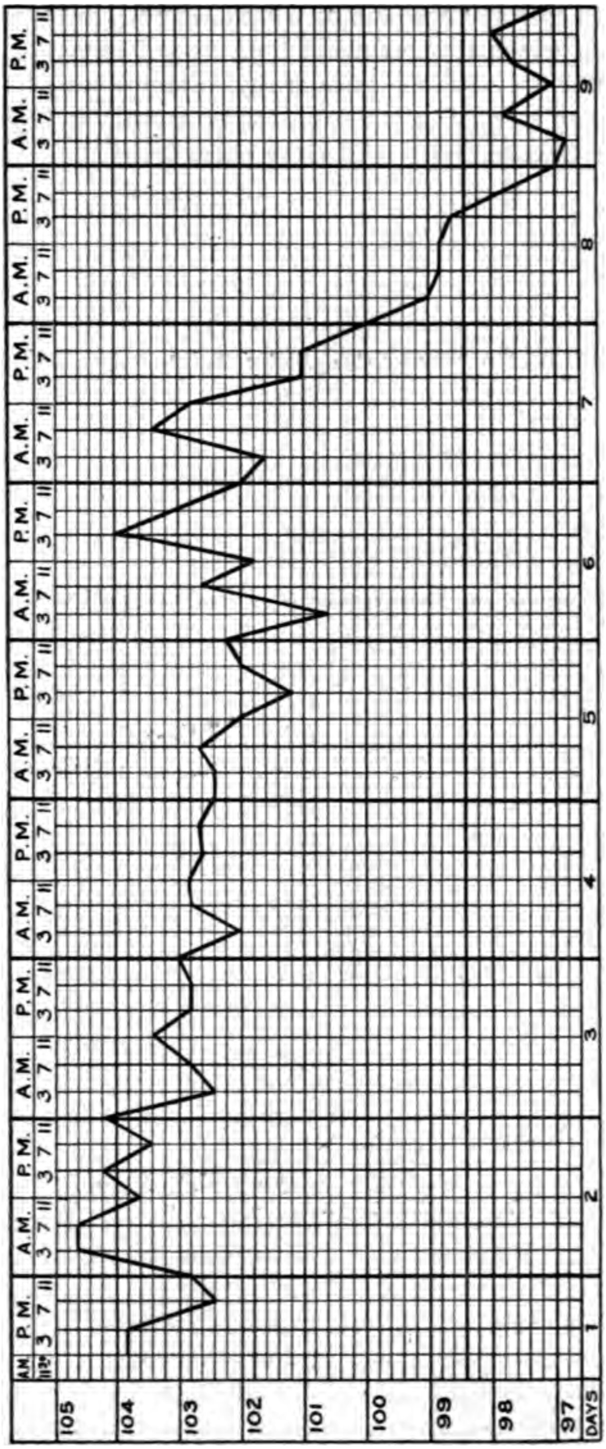
The crisis is sometimes reached at an unusually early date. I have seen it occur on the fifth day, and cases are on record in which it took place on the third day; but such are very exceptional.

High temperature does not have the unfavorable significance in croupous pneumonia that it has in numerous other diseases. Thus, recoveries from attacks in which the temperature reached 107° F., or more, are not very uncommon. On the other hand, many of the fatal cases are characterized by low fever throughout the entire course of the disease.

It is an interesting fact that the febrile course is not coincident with the pathological changes as indicated by the physical signs. Fever is a prominent feature before physical exploration can possibly afford a single sign of its cause; and after the disappearance of the fever by crisis, the evidences of pulmonary consolidation continue for quite a period without any abatement.

The febrile phenomena of *catarrhal pneumonia* are very different from those of the croupous variety. The temperature may rise either rapidly or gradually, but rarely to a point above 103° F. Chill may be present, but is an unimportant diagnostic factor. The morning remissions are more

FIG. 12.



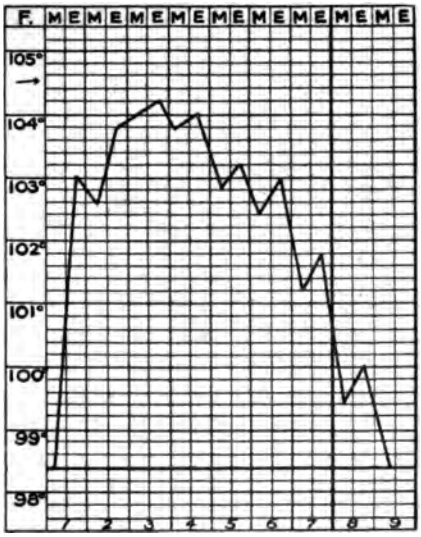
Illustrating pneumonic fever temperature-curve.

marked than in croupous pneumonia. The fever pursues no definite course, and disappears, after a long or short time, by lysis, with the relief of the pulmonary condition. Afebrile cases are by no means uncommon, although this fact seems to have failed of recognition by authorities in general. The diagnosis in such cases is based upon the physical signs.

5. Scarlatina; Scarlet Fever.

In scarlatina, the fever rises suddenly to a considerable height (104° F., or more) on the first day. In fact, it may be said to be the only contagious disease of childhood characterized by sudden accession of fever without preliminary ill-health. The characteristic eruption appears on the second day, the fever continuing until the fading of the cutaneous manifestations, declining *pari passu* with the eruption. The normal temperature is reached by the seventh or eighth day. The departure of the temperature-curve from the orthodox type just outlined, or its failure to attain the normal standard by the seventh or eighth day, is strongly suggestive of the presence of some complication, usually otitis media, lymphatic inflammations or acute nephritis. In severe cases, of course, the fever may remain at the maximum for several days; or, indeed, it may never show any tendency to abatement, owing to the steady progress of the malady to a fatal termination. In mild cases the fever may be very slight, or even absent. As a rule, the intensity of the fever is a fair guide as to the severity of the case. Still, it must be remembered that some very malignant cases are associated with but slight rise of temperature.

Fig. 13.



Temperature-curve of scarlatina.

In addition to the fever, the ordinary typical case of scarlatina presents the following symptoms: (a) The rash; (b) sore throat; (c) strawberry tongue; (d) various nervous and constitutional phenomena.

Malignant and complicated cases present the following: (*a*) Acute parenchymatous nephritis; (*b*) otitis media; (*c*) arthritis; (*d*) endocarditis; (*e*) cellulitis and glandular abscesses; and (*f*) pleurisy.

Taking, first, the symptoms of typical cases, the (*a*) **rash** constitutes a prominent feature. It appears on the second day of the disease—occasionally within twenty-four hours of the onset of the fever. It is first manifested usually about the chest, and is made up of two elements, namely, a general erythema of the affected parts, to which are added numerous fine, reddish dots. Pressure causes the disappearance of the redness, which returns as soon as the pressure is relieved. As a rule confluent, it sometimes assumes a patchy form, intervals of normal skin intervening between the parts covered by the eruption. The color is usually a bright red. From the chest it spreads until it invades the entire body, being fully developed within forty-eight hours. It begins to disappear in the course of from four to six days, no redness remaining by the ninth day. It is followed by desquamation, which takes place in the shape of fine scales. When the eruption has been severe, the epidermis is exfoliated in large flakes.

(*b*) **The Sore-Throat.**—The involvement of the throat is evident almost from the onset of the fever. The objective appearances indicate that this symptom is the mucous membrane prototype of the cutaneous manifestations. In mild cases there exists only a bright redness of the pharyngeal mucous membrane, with or without swelling of the tonsils. In those of more severe grade, there are swelling and infiltration of the parts, simulating the phenomena of follicular tonsillitis. In still more severe cases the appearances are those of a membranous angina, leading to the suspicion that diphtheria has appeared as a complication. Glandular involvement is common.

(*c*) **The Strawberry Tongue.**—In the beginning the tongue is heavily coated with a thick white fur; but this peels off in the course of two or three days, leaving the organ a bright-red color. The fungiform papillæ, being enlarged, gives the tongue the appearance which has not inaptly been compared to that of a strawberry. This condition is regarded as an important diagnostic symptom, as it occurs in no variety of sore-throat other than that of scarlatina.

(*d*) **Nervous and Constitutional Symptoms.**—Owing partly to the effect of high fever upon a young subject, and partly as the result of the toxæmia, nervous symptoms are ofttimes prominent in scarlet fever. Headache is nearly always present. In mild cases there may be nothing, in addition, except a slight restlessness. In those of a more severe type delirium occurs at night, and at the height of the disease may be constant. The onset of the disease is sometimes associated with convulsions. The latter symptom, occurring at this time, is not to be viewed with any alarm. Convulsions occurring late in the course of the disease are generally due to uræmia, and should be regarded as a very serious matter. They may also be due to hyperpyrexia and profound toxæmia.

The ordinary typical scarlatina, while a disease to be respected, is nevertheless one offering a favorable prognosis, excepting in very young children. When, however, the case is one of the malignant variety, or complications appear, the outlook is serious.

(*a*) **Variations in the Classical Symptoms.**—The constitutional symptoms may be unusually severe from the first. In one type of cases,—rarely observed at the present day,—the patient becomes delirious at once; this passes rapidly into coma or stupor, and the patient dies from the intensity of the toxæmia before the characteristic symptoms have had an opportunity to develop. In another class of cases the fever is unusually high, and all the objective signs are severe. Departures from the typical temperature-curve include persistence of the fever beyond the orthodox period for defervescence, or the substitution of the continuous type of fever by that of a markedly remittent type dependent upon a septic process or the advent of some suppurative lesion.

The **rash** may appear in patches, and instead of presenting the characteristic "boiled-lobster-like redness" is of a dusky hue, and it may present numerous small elevations.

The **throat symptoms** may present numerous departures from the ordinary erythema. Thus there may be, as already stated, a membranous formation. In some cases it consists of a simple exudate; in others it is a superficial sloughing. It occasionally happens that scarlatina and diphtheria coexist. The diagnostic difficulties attendant upon this state of affairs can only be surmounted by a bacteriological examination of the exudate, studied in connection with the rational symptoms. One of the varieties of malignant scarlatina is attended by extensive ulceration of the throat (*scarlatina anginosa*). The resulting destruction of tissue may impair greatly the functional integrity of the parts.

These anomalous pathological changes in the throat may extend to the nasal cavities, producing rhinitis, with purulent discharge.

(*b*) **Acute Parenchymatous Nephritis.**—In this lesion we have a complication for which the attendant should ever be on guard, not only because of its menace to life, but also because of the possible sequences. Albuminuria is so frequently observed in the course of scarlatina that it may well be regarded as one of the regular symptoms of the disease. A true nephritis is sufficiently rare to be accepted only as a complication. The majority of cases are believed to occur in from the second to the fourth week of the disease. The outbreak of nephritic symptoms is preceded by the urinary symptoms of acute nephritis, including albuminuria, scanty and smoky urine of high specific gravity, and containing epithelial, granular and blood-casts, blood-corpuscles, etc. If, however, the state of the urine is persistently disregarded, the nephritis may not be suspected until the advent of dropsy or the ordinary phenomena of uræmia.

(*c*) **Otitis Media.**—Inflammation of the middle ear is the result of infection of that structure by reason of the extension of inflammation from the throat. Its onset is characterized by earache. In the course of a few days spontaneous rupture of the membrana tympani takes place, with relief of the pain and a free discharge of pus. In some cases, unfortunately, the inflammation extends to the mastoid cells, in which case rupture does not bring with it the customary relief from pain. Under wise remedial measures it is usual for the otitis media of scarlatina to clear up rapidly. Neglect may lead to chronic otorrhœa.

(*d*) **Arthritis.**—The arthritic complications of scarlatina will be described in the chapter devoted to the study of joint-lesions. It is sufficient to note, at this time, that the joint-lesions are not rheumatic in origin, as claimed, but are septic.

(*e*) **Endocarditis.**—This complication is not very uncommon, but is apt to be overlooked. It is nearly always of the simple variety, and may give rise to permanent valvular lesions. Pericarditis with either sero-fibrinous or puruleut effusion is sometimes observed. Heart complications are of especially frequent occurrence in cases attended by arthritis or nephritis.

(*f*) **Cellulitis and Glandular Abscesses.**—Nearly all cases of scarlatina are attended by some glandular enlargement, which subsides with the disease. When sepsis is well-marked, the affected glands may become severely inflamed and undergo suppuration. It may even happen that the peri-glandular tissues are involved; in which case the affected parts—usually the neck—become swollen and brawny, and, if proper surgical treatment is not instituted at once, extensive suppuration and sloughing ensue.

(*g*) **Pleurisy.**—Pleurisy is a rare complication. It is apt to occur during convalescence. The associated effusion is purulent, as a rule.

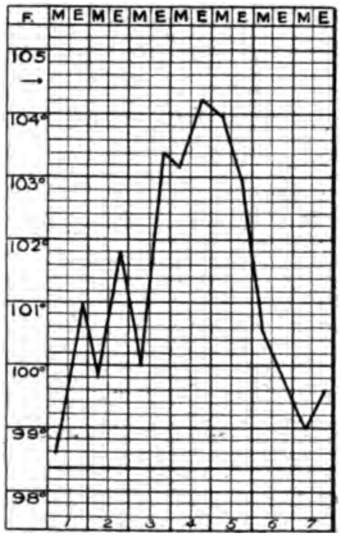
Sequelæ.—Scarlatina is apt to be followed by numerous sequelæ. Some of these are the continuance of one of the complications to which reference has been made; others include chorea, cerebral, spinal and peripheral paralyses, cancrum oris, gangrene, etc.

The possibility of mistaking scarlatina for other diseases does not appear to be very great; and yet such errors do occur, the conditions causing confusion, including measles, diphtheria, septicæmia and rötheln. If the observer but bear in mind the characteristic phenomena of scarlatina, and their sequences and relation to each other, mistakes will be almost impossible.

6. Measles.

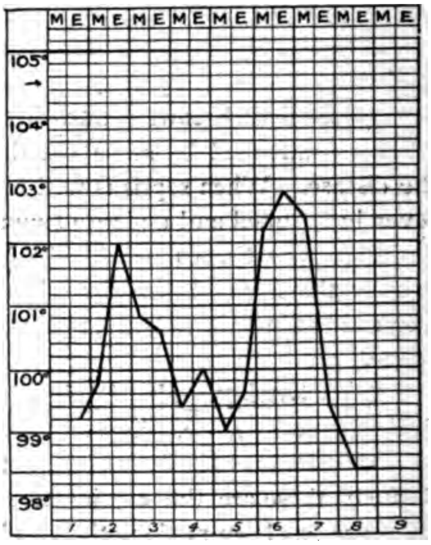
Measles is characterized by more than one type of temperature-curve. In one there is an initial rise of temperature to 103° F. or thereabouts, falling promptly to a little above the normal. It is in exceptional instances only that this preliminary fever is observed by practising physicians, by reason of not having been called sufficiently early. On the fourth day there is a fresh accession of fever, which may be as high as 105° F., and at the same time the eruption appears—the maximum of fever corresponding to the full development of the rash. It ordinarily remains at the maximum until the time the eruption begins to fade, which is not longer than two days, and usually not more than one, when it falls gradually during a period of twenty-four to thirty-six hours. In another type the fever rises gradually, with morning remissions and evening exacerbations, until the appearance of the eruption, at which time it is at its height. It then follows the curve of defervescence, as above described. The persistence of fever beyond the time specified suggests the presence of some complication, which in measles is usually bronchial catarrh, broncho-pneumonia, gastro-intestinal catarrh, or otitis media. The remarkable similarity of the rash of rötheln to that of measles may give rise to diagnostic errors, but the thermometer should prove a valuable means of differentiation. In rötheln there is no preliminary fever, and the rise of temperature lasts but twenty-four hours, if, indeed, it continues for even so long a time. Other than this, the temperature-curve of measles cannot be said to possess a positive diagnostic value. Departures from the types outlined are, however, significant as sounding the onset of a complication.

Fig. 14.



Temperature-curve of case of measles.

Fig. 15.



Temperature-curve of case of measles with preliminary fever.

Associated with the fever in measles, and constituting important clinical features of the disease, are catarrhal inflammation of various mucous membranes, but especially those of the eyes and the upper respiratory tract, and the eruption.

The **catarrhal symptoms** are first manifested by coryza and lachrymation, which appear with the fresh accession of fever on the fourth day of the disease. They are followed within twenty-four hours by a bronchitis attended by a cough, which to the ears of the experienced is in many instances characteristic. This cough constitutes the chief source of annoyance in most cases. Catarrhal involvement of the digestive tract is exhibited by diarrhœa.

The **eruption** appears generally on the fourth day; sometimes it may be delayed to the sixth. It is first observed on the forehead, whence it spreads to the balance of the face. On the following day it will have involved the trunk and extremities. The rash itself consists of numerous papules, arranged, for the most part, in crescentic patches. In color they are of a rather dusky red. With the exception of cases attended by hæmorrhagic extravasations, the rash disappears momentarily under pressure of the finger. The rash and the fever begin to show a marked abatement on the sixth day, unless some complication is present, in which case the fever is apt to continue.

Even before the onset of the eruption, evidences of measles will be found in the mouth in the shape of the lesions known as *Koplik's spots.* (*Vide* chapter on Mouth Symptoms.)

The uncomplicated case of measles as above outlined is rarely a serious disease, practically all cases making an uneventful recovery. Sometimes, by reason of factors which are not well understood, the disease does not run a benign course, or is attended by complications which endanger life or interfere seriously with the functions of important organs. It is believed that the tubercular diathesis and bad hygienic surroundings are the principal causes of these unfortunate cases. Of the complications, catarrhal pneumonia is most frequently observed, and is, moreover, the most dangerous. The conjunctival inflammation sometimes persists as phlyctenular or scrofulous ophthalmia. Otitis media, with catarrhal discharge, is rather common. The affections of the special sense-organs sometimes result in permanent damage to the affected parts. Patients who have just recovered from measles seem to offer an especially favorable soil for tubercular infection.

Cases of measles departing from the normal type have been favored with special designations. Thus, the catarrhal symptoms may be absent, and we speak of *morbilli sine catarrho;* the eruption may be absent, and then it is known as *morbilli sine exanthemate;* the toxæmia may be very profound, and the patient is overwhelmed with the intensity of the poisoning, in which case it is *morbilli siderans;* and finally we have *morbilli nigra,* or black measles, in which hæmorrhages take place from the mucous membrane and into the cutaneous structure.

7. Rötheln; Rubella.

Rötheln is a contagious disease, at one time believed to be a hybrid of scarlatina and measles. The fever, always of a mild type, appears within a few hours after the prodromal symptoms, which are always mild. The fever rarely rises to 101° F., and usually not above 100° F. The rash appears the first day; its advent may, however, be delayed until the second day, but never longer. It consists of bright rose-red, slightly-raised papules, more or less scattered, but which may become confluent. Sometimes the rash presents the bright red-punctate appearance characteristic of scarlatina. Glandular enlargement, especially in the post-cervical region, is noticed very early in the course of the disease. Soreness of the throat, with redness of the pharyngeal mucous membrane and swelling of the tonsils, is common; in this particular it presents a marked difference from measles. Constitutional symptoms other than the fever are absent. The rash and fever disappear about the end of the third day. The prognosis of rötheln is favorable. Complications and sequelæ are rare. Those which have been reported include tonsillitis, bronchitis, pneumonia, albuminuria, and adenitis.

8. Variola; Small-Pox.

Variola is possessed of a temperature-curve subject to fewer departures from the characteristic type than any of the acute infectious group. As in measles, there is an initial rise of temperature,—often to a high point (105° F.),—but unlike measles in its association with severe constitutional symptoms, as backache, headache, and severe prostration. On the second or third day the temperature falls,—almost to the normal point, in some cases,—and at the same time the eruption appears. Herein is found a marked difference between small-pox and all other of the exanthemata,—*the eruption appearing while the fever declines.* Attention to this one point will save many a mortifying diagnostic error. The temperature remains slightly above the normal until the ninth day, at which time the contents of the vesicles undergo purulent transformation, when the fever rises to as high or a higher point than before. This is referred to as the secondary or suppurative fever. It continues for about a week in uncomplicated or favorable cases, then disappearing by lysis.

Departures from this standard must be observed as follows: (i) In *varioloid* or mild cases, in which the secondary fever is slight or practically absent; (ii) in severe cases, in which the temperature does not fall to the normal on the appearance of the eruption, or the period is marked by evening rises of temperature. The secondary fever is more persistent, and in fatal cases continues at a high point until death.

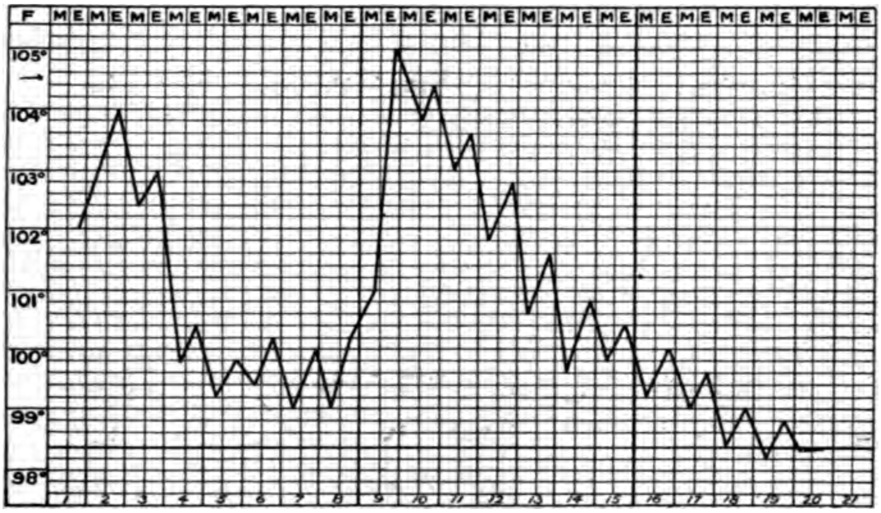
The symptoms of small-pox other than the fever include headache, backache, and the eruption.

(*a*) The **headache** appears almost at the same time as the onset of the fever. It is remarkable for its severity and its constancy.

(*b*) The **backache** is likewise an early and severe symptom. It has long been regarded as an important diagnostic aid, especially in the presence of an epidemic. Both it and the headache disappear with the appearance of the eruption.

(*c*) The **eruption** makes its appearance on the third or fourth day. At first it consists of macules, which are soon transformed into papules. They are usually found first on the forehead, spreading later to the balance of the face, and to the trunk and extremities. When the lesions are at all numerous, and close together, they may simulate the eruption of measles. Palpation gives the impression that a considerable thickness of the skin is involved in the infiltration; hence the well-known comparison to "shot beneath the skin." On the second day of the eruption vesiculation commences, and is completed in twenty-four hours. Next the vesicles become umbilicated. On the eighth or ninth day the contents of the vesicles become puriform, and the cutaneous surface surrounding the lesions becomes inflamed, and even œdematous. About the eleventh or twelfth day the pustules rupture and form crusts. At this time cutaneous inflammation subsides rapidly, and healing progresses. Finally the crusts drop off, leaving a reddened, cicatricial depression. With time, this changes to a white "pit," which in severe cases is permanent. The cutaneous lesions may make their appearance upon the various mucous membranes, and thereby give rise to symptoms varying according to the functions of the

FIG. 16.



The typical temperature-curve of small-pox.

part affected. The lesions of the mouth and throat cause only local discomfort, and interference with mastication and deglutition. The laryngeal lesions give rise to local suffering and cough, and in some cases, by reason of the production of œdema, serious interference with respiration. The appearance of the eruption on the conjunctivæ and corneæ may prove to be a serious matter, for they may be followed by perforation of the cornea and destruction of sight; or the resulting cicatrices may produce permanent corneal opacities.

The symptoms above described are those of the ordinary type of the disease, *i.e.*, *variola discreta*. *Variola confluens* is a more severe variety. Its constitutional manifestations are much more marked, and the lesions are so closely placed as to be confluent. The eruption, moreover, appears more promptly than in the discrete form, sometimes as early as the second day; and instead of the macules appearing in gradual succession on various portions of the body, commencing with the face, the entire cutaneous surface is affected simultaneously. This variety of small-pox presents a most serious prognosis, more than half of the cases dying.

Variola hæmorrhagica, "black small-pox," differs from the varieties already described in the appearance of hæmorrhage in the lesions. This complication may manifest itself at any stage of the disease—the earlier, the more unfavorable the outlook. When the hæmorrhages take place into the papules, the condition is known as *purpura variolosa*, and this generally has a fatal result within a few days.

In **varioloid,** or small-pox which has been modified by vaccination, the lesions may be few and far between, and the constitutional symptoms correspondingly mild. It is in cases of this class that diagnostic errors are most frequently committed, the disease passing unnoticed or being mistaken for chicken-pox.

Nothwithstanding the well-defined symptomatology of small-pox, diagnostic mistakes are common. This arises not so much from actual ignorance as from the fact that the widespread dread of the disease and the fear of falling into error cause temporary deficiencies of judgment. Measles seems to be the disease responsible for the most "breaks." In discrete variola there seems to be no possibility of confounding the diseases on their cutaneous manifestations. In the case of variola confluens, the eruptions in their incipiency may, to the inexperienced, bear a superficial resemblance. But a comparison of the associated symptoms helps to a correct conclusion. Measles does not have the severe invasion symptoms observed in small-pox, and the fever is associated with well-defined catarrhal symptoms. The subsidence of the fever with the appearance of the eruption is of the highest importance in pointing to small-pox. Moore's "grisolle sign" may prove extremely useful: "If, upon stretching the affected portion of the skin, the papule becomes impalpable to the touch, the eruption is caused by measles;

if, on the contrary, the papule is still felt when the skin is made tense, the eruption is the result of small-pox."

The differentiation of variola and *varicella* is a more difficult matter, and mistakes are common. Varicella presents few or no constitutional symptoms at the onset. The eruption appears first about the trunk and on the scalp. The lesions come out in successive crops. The initial macules or papules are transformed into vesicles within a few hours after their first appearance. They do not become umbilicated. The fever subsides before the appearance of the rash.

The number of diseases which have been confounded with small-pox is surprisingly large, and it is impossible to lay down a prior data for their differential diagnosis. Some queer problems sometimes come up, *e.g.*, the differentiation of small-pox and a camphor rash resulting from the taking of a diarrhœa mixture.

The complications of small-pox are few, and relate almost exclusively to increased severity of special symptom-groups. The sequelæ include, aside from the permanent effects of the lesions, numerous nervous manifestations, as epilepsy, multiple neuritis, vascular degenerations, etc.

9. Vaccinia.

Following vaccination, it is not very uncommon for a successful "take" to be attended by high fever, 104.5° F. This subsides almost at once, however, so that the normal is reached inside of forty-eight hours.

10. Varicella; Chicken-Pox.

Fever does not seem to be a very important symptom of varicella; indeed, it may be so slight as to escape notice. Usually it is of short duration. Occasionally, in debilitated or highly neurotic children, it may reach a high point. Thus I have observed it at 104° F. in two children previously weakened by whooping-cough.

Constitutional symptoms are rarely present. The rash is the important clinical feature. It makes its appearance within twenty-four hours of the onset of the fever, and it first invades the trunk or extremities. The initial lesion is a papule, which does not infiltrate the parts as deeply as do the lesions of small-pox. In the course of a few hours the papulæ become transformed into vesicles, and in thirty-six to forty-eight hours more into pustules. An important feature is the appearance of the lesions in successive crops, so that it is not uncommon to observe present, at the same time, lesions in various stages of development. As a rule, the pustules heal without pitting; but sometimes one or more marks may remain permanently.

The complications of varicella include noma, gangrene and nephritis.

11. Erysipelas.

The characteristic of the febrile movement of erysipelas is an initial chill or rigor, followed by a sharp rise of temperature, usually to a high point, and often to 105° or 106° F. Defervescence may take place by either lysis or crisis, but usually by the latter.

Very shortly after the onset of the fever the local lesions appear. In the majority of cases it is the face or adjacent parts that are involved. The appearances will vary according to the extent of the infection. If the lesion is superficial (*cutaneous erysipelas*), there will be marked redness, heat, and some swelling. The latter condition is shown by slight œdema and the elevation of the affected part above the surrounding healthy skin. The line of demarcation between the affected and unaffected parts is well-defined. In some cases the inflammation spreads from part to part, each fresh involvement being characterized by a temperature flight. When the deeper structures are also invaded (*phlegmonous erysipelas*), the cutaneous surface also presents a red, inflamed appearance, but the extensive infiltration imparts to the lesion a boggy feel.

In either variety of erysipelas there may follow vesiculation over the affected area. The blisters may coalesce and form large blebs, the contents of which may become puriform. They usually rupture and form crusts. During convalescence, the epidermis over the parts which have simply been inflamed is exfoliated in large pieces.

In the phlegmonous variety, suppuration of the underlying tissues not infrequently takes place, leading to abscesses and sloughing.

Very serious complications of erysipelas are found in the so-called metastases, by which we refer to infection of the various serous membranes. Thus it is that we have presented the symptoms of meningitis, pleuritis, peritonitis, pericarditis, etc. Many of the cases of so-called meningitis associated with erysipelas are undoubtedly the result of the profound toxæmia and the high fever upon the brain.

The constitutional symptoms are always well-marked, especially in persons whose constitutions have been undermined by advancing years, renal disease, and alcoholic indulgence. These symptoms include digestive disturbances, delirium, dry and brown tongue, and great prostration.

The cause of erysipelas being an infection of the skin by the streptococcus pyogenes, ofttimes through an operation wound, a history to that effect aids in the diagnosis.

Erysipelas is always to be regarded as a serious disease. The average mortality is about 10 per cent. It is impossible to formulate a prognosis *a priori* in any individual case, as it is always uncertain whether or not the inflammation will spread to other parts.

12. Tuberculosis.

Fever may be present or absent in tuberculosis. When present, it is the result of microbic infection, *i.e.*, of the constitutional effects of the tox-albumins generated by the bacilli of tuberculosis and suppuration. In many cases it is, according to Leyden, strictly tubercular, as demonstrated by the presence of tubercle bacilli and the absence of the micro-organisms of suppuration—the staphylococci and the streptococci. In cases in which well-defined cavities exist, the suppurative process is added to the tubercular as a fever-producing factor. In the majority of cases the temperature-curve of pulmonary tuberculosis corresponds closely with that of septicæmia, although not usually attaining as high a point as in the latter disease. The fever may be continuous, remittent, or intermittent. In the early stages it is not uncommon to find a morning subnormal temperature and an evening febrile rise; the latter, however, rarely being of a high grade. Such a condition if at all persistent, and especially if associated with symptoms denoting deterioration of the general health, may be regarded as suggestive if not diagnostic of incipient tubercular disease of the lungs, physical signs not being attainable owing to the deep situation or the small size of the lesions. Ordinarily, the activity of the febrile movement is directly proportionate to that of the pathological process, as evidenced by subjective and objective symptoms. Exceptionally, however, the fever associated with vague feelings indicative only of a sense of illness constitute the sole manifestations of the disease, thus leading to the mistaken diagnosis of typhoid fever or malaria, according to the type of fever present. The maximum temperature may attain any point between 101° and 106° F. The regular use of the thermometer in tubercular cases is especially important, because activity of fever suggests activity of lesions; and, again, because measures directed against the fever certainly control the general condition, making the patient more comfortable and prolonging his life.

It is in acute miliary tuberculosis that fever constitutes an important symptom. In these cases, physical exploration of the chest, even in the hands of the experienced, is unsatisfactory as to its results; hence the great liability to diagnostic error. The fever presents no settled type, although usually described as continuous. This last-mentioned fact renders its confusion with typhoid fever a very easy matter; in fact I might say that such mistakes are exceedingly common, if not altogether excusable. The tubercular fever is associated from the beginning with an exceedingly rapid pulse, even as high as 150 per minute,—a condition that may be safely stated as always absent in typhoid fever, excepting in the terminal stages. While the pulse-rate falls with the subsidence of the fever, it nevertheless remains high. While not of diagnostic value, it is important to note that the fever of tuberculosis is associated with profuse and exhausting perspiration.

The fever of acute miliary tuberculosis may also be mistaken for that of meningitis. Here, again, the character of the pulse affords a tolerably safe diagnostic guide, being rapid in tuberculosis, and slow or but moderately increased in meningeal inflammation.

Fowler, while referring to the existence of a continuous fever in the course of acute miliary tuberculosis, does not lay the stress on it which its importance demands. To my mind, the temperature-disturbance is of the highest importance from both diagnostic and prognostic standpoints. These cases are very apt to escape observation by lung specialists, for they are too frequently diagnosed as typhoid fever. Fowler also refers to an inverse type of fever, which he says "occurs in cases of pulmonary tuberculosis previously of the chronic variety, and also in cases of general acute tuberculosis, and may then be the only trustworthy sign of the nature of the disease."

Fowler's conclusions respecting pyrexia in the course of pulmonary tuberculosis are, however, of great practical value, and are as follows: "(1) The degree of fever present in cases of tuberculosis is a measure of the activity of the morbid process. (2) Marked pyrexia is always associated with an increase in the tubercular lesions. (3) The action of the septic organisms present in the lung has little or no share in its production. (4) The occurrence of miliary, fibroid and caseous lesions is indicated by distinct types of pyrexia."

The third conclusion can hardly receive general acceptance, for there are undoubted cases in which the suppurative process is a most important factor in the production of pyrexia. His fourth proposition is especially interesting. *Caseous tuberculosis* is characterized by a high remittent type of fever, which in the acute cases runs throughout the entire course of the disease. He claims that a higher temperature is attained than in miliary tuberculosis. Later, when cavities form, and vital power is diminishing, the fever tends to assume the intermittent form.

In *chronic fibro-caseous tuberculosis* the temperature is characterized by great variety of range. When the lesions are quiescent, fever is usually absent; when they are aroused to activity, fever of a type appropriate to the conditions present appears.

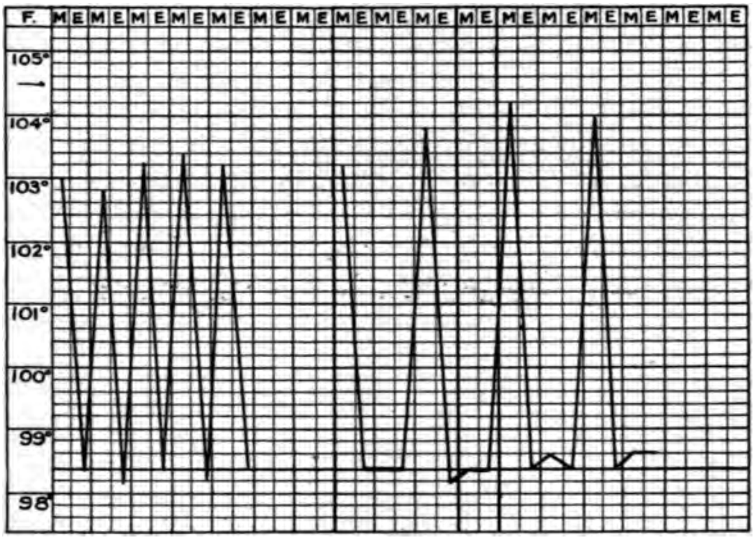
Fibroid tuberculosis has almost complete absence of fever. Indeed, the temperature may be subnormal for days at a time.

13. Malarial Fevers.

The typical malarial fever adopts as its characteristics periodicity and intermittence. The fever recurs every first, second or third day at approximately the same hour, and in each paroxysm follows a definite course. When occurring daily, we speak of the fever as *quotidian;* when on alternate days, *tertian;* and when every third day, *quartan.* These various types of periodicity are dependent upon parasitic causes. Three varieties of malarial organisms have thus far been recognized, namely, the tertian, the quartan, and the æstivo-autumnal parasites. The tertian and the quartan produce the types of fever after which they are named, while the æstivo-autumnal is the cause of the various pernicious malarial fevers. The quotidian fever is the result of infection by two sets of tertian parasites, each set developing on alternate days, and each producing its febrile paroxysm. A quotidian fever is therefore in reality a double tertian.

To return to the temperature-curves of malaria: The characteristic, as already stated, is intermittence and periodicity in the typical cases. The fully-developed paroxysm consists of three stages, namely: (*a*) chill, (*b*) fever, and (*c*) sweat. The chilly stage is occasionally absent. Sometimes it consists of but slight shivering or subjective sense of coldness. In the majority of cases it is well marked, the shiverings and shakings being violent. The patient's sense of coldness is so great as to demand that he be well covered with several thicknesses of blanket. The surface of the body may be cold and cyanotic, at the same time the thermometer shows that there is an actual rise of temperature. The skin exhibits the phenomenon known as "goose flesh," or *cutis anserina.* The stage of chill usually lasts from ten minutes to one hour. Then comes the stage of fever. The patient no longer requires to be covered. The skin becomes hot and dry. The thermometer shows a rapid rise of temperature, the maximum (usually 104° to 106° F.) being attained within a few hours. Then comes defer-

FIG. 17.



Illustrating the temperature-curves of quotidian and tertian types of malarial fever.

vescence, which takes place rapidly (though not as rapidly as the onset), and is accompanied by a profuse sweat. The entire paroxysm has a duration ranging from three or four to twelve or fifteen hours. The sweating is generally profuse,—sufficiently so to thoroughly soak bedding and clothing.

The typical paroxysm, as above described, recurs at a definite interval, this interval depending upon the particular case. Ordinarily the onset is observed in the morning, but cases beginning in the afternoon or at night are occasionally met with. The paroxysms may recur on the appointed days at precisely the same hour, or at an earlier (anticipating), or at a later (retarding) hour.

In the quotidian, or, more scientifically speaking, the double-tertian type, careful observation generally demonstrates similarities in the temperature-curves of alternate days,—thus indicating, without the aid of blood-examinations, the dependence of the attacks on the two sets of alternate days upon separate sets of organisms.

The quartan type of fever differs in its clinical phenomena from that of the tertian, already described, only in the fact that the paroxysms recur every third day. The presence of two or three sets of parasites, as in the case of the tertian, leads to increased frequency of paroxysms. Thus, with two sets in the blood, the fever appears on two successive days and is absent on the third; with three sets present, the fever appears daily,—quotidian dependent upon the quartan parasite—triple quartan intermittent fever.

The *æstivo-autumnal* types of malarial fever differ from those already described in the irregularity of their manifestations, dependent upon the fact that their special parasites do not appear in the blood in large, sharply-defined groups, and that they present marked variations in the cycles of their existence. In the early stages of these fevers some periodicity is manifested; and during this period the parasites develop in groups. It is obvious, therefore, that æstivo-autumnal malaria may present a quotidian, or tertian intermittent a remittent or a continuous type of fever. The paroxysms are usually longer-lasting than in the quartan and tertian types already described. The effects of the disease upon the patient are also more severe,—hence the names *pernicious* and *malignant intermittents*,—owing to the fact that the organisms are found in greatest numbers in the blood of the deeper viscera. The rise of temperature is but infrequently preceded by a chill, and takes place quite gradually. Marchiafava and Bignami assign a definite curve to the æstivo-autumnal fever, including a more or less sudden onset of symptoms, a pseudo-crisis, a pre-critical elevation of temperature which often reaches a higher point than has been previously attained, and finally the actual crisis. If such a characteristic curve exists, it is certain that the atypical cases are very common.

Malignant intermittents are prone to merge into malignant remittents by anticipations of the paroxysms, one attack coming on before another has entirely subsided. Even a continuous fever may be thus produced, although careful thermometric observations give indications of the presence of a paroxysmal course. Sometimes, however, the paroxysmal course of the fever is completely hidden, in which event the cases bear a perplexing similarity to one of typhoid fever. Under these circumstances the problem can be solved only by a microscopic examination of the blood.

Thayer thus sums up the modifications which the temperature-curve of æstivo-autumnal fever may undergo:

"(1) Through modifications of the curve in the individual paroxysm: (*a*) The lack of a sharp initial elevation, so that the curve rises in a slow and continuous manner; (*b*) the occurrence of a pseudo-crisis, so that the attack tends to lose its individuality; (*c*) the prolongation of the paroxysm, which is usually associated with an exaggeration of the thermic oscillations during the fastigium.

"(2) The modifications in the succession of the paroxysms may be (*a*) the retardation of the paroxysms; (*b*) the prolongation of the paroxysms, by which apyrexia is made incomplete; (*c*) the occurrence of slight oscillations in temperature during the period which ought to be one of complete apyrexia; (*d*) the reduplication of the attack."

The symptoms associated with the pyrexia in malarial fevers present the greatest variations not only as to the different varieties of infection, but also as to the cases of the same form of the disease. Much depends upon the extent of infection, the general health of the patient, and the promptness with which proper treatment is instituted. The early enforcement of proper therapeutics should rob the disease of all constitutional symptoms belonging to the malarial cachexia.

In the beginning of cases of malarial intermittent fever, the only symptoms during the paroxysms are such as we ordinarily expect to find in association with rigors and pyrexia of like degrees of severity. During the interparoxysmal period the patient complains of great prostration, together with a number of subjective symptoms of indefinite character. Physical examination reveals some enlargement of the spleen, which, in neglected cases, may become quite marked.

The æstivo-autumnal malarial fevers present well-defined constitutional symptoms; and this remark applies to both the remittent and the pernicious forms of the disease. In the remittent or continued malarial fever the symptoms may closely simulate those of typhoid, owing to the low delirium, the dry brown tongue, the great prostration, and the splenic enlargement. An associated hæmatogenous jaundice speaks in favor of malaria as opposed to typhoid fever; but the crucial diagnostic test is found in the effect of quinine, which will produce marked relief of symptoms in the malarial cases. The blood examination also gives diagnostic information, discovering the æstivo-autumnal parasite in the case of one disease, and the Widal reaction in the case of the other.

Much confusion has existed for years respecting the diagnosis of the fevers current in tropical and sub-tropical regions. It seems to me that the effort to differentiate many types is a waste of valuable time, and that the position assumed years ago by Goodno, and later by Osler, that there are but two forms of continued fever existent—one due to typhoid and the other to malarial infection—is the correct one.

The pernicious malarial fevers present a symptomatology that is not liable to be confounded with that of typhoid fever. The cases of this affection may be divided clinically into three types : (*a*) *The comatose*, characterized by onset, with symptoms of profound cerebral disturbance, as delirium and coma, associated with high fever. The patient may die within a few hours, or he may regain consciousness, only to perish in the next paroxysm. (*b*) *The algid form*, characterized by a sudden onset, as in the form just described; but the symptoms relate to the gastro-intestinal tract, and include nausea, vomiting, and great prostration. While the patient may complain of feeling warm, the temperature is actually subnormal. The evidences of failing circulation are well-marked. (*c*) *The gastro-enteric form*, in which vomiting and purging are the prominent symptoms. The discharges may present some resemblance to those of cholera. Ofttimes the stools are blood-tinged, or they may consist of pure blood. The patient generally dies in a few hours, in collapse. (*d*) *The hæmorrhagic form* is observed only after the malarial infection has continued for a considerable period of time. The bleeding takes place from the various mucous surfaces. These cases are apt to be associated with profuse sweats, which of themselves are a source of danger, by reason of the great prostration which they produce.

The symptoms noting a chronic malarial cachexia are but few in number. They include anæmia, enlargement of the spleen, hæmorrhages, occasionally jaundice, and irregular fever. This list of symptoms is infinitesimally small, when viewed in comparison with the host of phenomena which one so frequently hears assigned to malarial poisoning. As a matter of fact, true malaria is sufficiently rare to be a curiosity, providing proper treatment has been instituted. Cases in which this condition is incorrectly diagnosed are as numerous as the leaves of the forest.

14. Dengue.

The fever of dengue is of rapid onset, and is associated with rigors, headache, backache, and general soreness. The symptom last named has led to the application of "break-bone fever." The maximum temperature is attained on the second or third day, when the temperature falls gradually, reaching the normal on or about the fifth day. There may follow a secondary fever of short duration, but which never attains the maximum height of the primary fever. The temperature in dengue may reach any point between 101° and 106° F.

The **rash** presents no diagnostic features. In the beginning there may be a rash which resembles that of scarlatina quite closely. It is, however, of but short duration. On about the fourth or fifth day of the disease there appears another eruption, which may be erythematous, urticarial or herpetic.

The only disease with which it seems possible to confound dengue is acute articular rheumatism. But dengue lacks the articular swellings, and the pains are diffused over numerous other portions of the body. Rheumatic fever does not present the eruptive feature of dengue.

In the presence of a yellow fever epidemic there may be diagnostic difficulties at the outset, but the nature of the case should not long remain in doubt.

The prognosis of dengue is almost invariably favorable.

15. Cerebro-Spinal Fever; Cerebro-Spinal Meningitis.

Though a frequent symptom of cerebro-spinal fever, fever is by no means a constant one, as it is often noteworthy because of its absence; and even when present, it cannot be said to have any diagnostic or prognostic value, because of the great variety of curves presented and the lack of relation between the degree of pyrexia and the ultimate result. The many complications and sequelæ make their impress upon the fever-curve of the primary disorder. Even though the latter be absent, no regularity is certain. Fatal cases have been known to run their entire course without fever on the one hand, and have been characterized by hyperpyrexia on the other. In the majority of cases the temperature rises on the first or second day to a moderate degree, *i.e.*, to 102° F. In fatal cases it may continue to rise for several days; in others it has remained slightly above normal until near the end, when hyperpyrexia suddenly supervenes. In favorable cases the fever disappears by lysis. In those pursuing a chronic course, *i.e.*, a month or more, there are usually periods of apyrexia, interrupted by short febrile paroxysms.

The symptoms associated with the fever are many, the chief of which relate to the nervous system and the skin. There are also disturbances of the pulse and respiration, the digestive apparatus, and the special senses.

The symptoms referred to the nervous system include headache, pain in the back and extremities, rigidity and contractures, convulsions, paralyses, delirium, and coma. Of all these symptoms, the headache is the most constant; indeed, it is never absent. It may invade any portion of the head, but involves the frontal and occipital regions more frequently than other parts. It soon spreads, however, to invade the entire cranium, and is then the cause of most excruciating suffering. The backache is fairly constant. It is generally experienced with the greatest severity in the cervical regions, but may invade other localities, or even the entire spine. It is greatly aggravated by any motion, and hence compels the patient to assume a state of complete immobility. The pains in the extremities are due to irritation of the posterior spinal nerve-roots, and are usually associated with muscular contractions. The rigidity of the spine may be sufficiently great to produce opisthotonos, and seems to occur independently of the pain. The rigidity of the extremities may come in paroxysms, and, when the muscles of the jaw are involved, may suggest the possibility of tetanus. Convulsions, when present, are usually epileptiform in type. Sometimes, owing to the peculiar distribution of the pathological changes, they are localized. The paralyses are decidedly irregular in their types. They are to be regarded as among the infrequent symptoms. Delirium and coma present no points to distinguish them from the same symptoms occurring in connection with other cerebral disorders.

The cutaneous lesions have led to the designation "spotted fever," so commonly used by the laity when speaking of this disease. They are by no means invariably present, nor do they exhibit some particular type, as in the acute exanthemata. The lesions observed include herpes, petechiæ and rose-spots. The herpetic vesicles differ in no particular from those observed in other diseases. They appear with greatest frequency upon the face, but may involve the surface of the trunk. They make their appearance from the second to the fourth day. The most characteristic of the eruptions of cerebro-spinal fever is the petechial blotches. These may be either discrete or confluent. They appear early in the disease, and especially frequent about the medium-sized joints. The rose-spots simulate the eruption of typhoid fever, but are relatively uncommon, as compared with the varieties already named.

The lesions of the eye are somewhat numerous, and include conjunctivitis, keratitis, iritis, irido-choroiditis and retinitis. The auditory lesions include suppurative disease of the labyrinth, semicircular canals and tympanum. These are very apt to cause permanent destruction of hearing.

The complications include, aside from the disturbance of the special sense-organs, pericarditis, nephritis, arthritis and abscesses.

The great variations in symptomatology and clinical course presented by cerebro-spinal fever have led to several classifications of types of disease. Of these, those deserving of especial mention are the following, their names indicating, with a fair degree of accuracy, their symptomatic peculiarities : (*a*) The foudroyant ; (*b*) the comatose ; (*c*) the inflammatory ; (*d*) the typhoid ; (*e*) the delirious ; (*f*) the abortive ; (*g*) the petechial.

The diseases with which cerebro-spinal fever may be confounded include typhoid fever, influenza and tuberculous meningitis.

Typhoid fever rarely presents as severe a type of headache as is observed in cerebro-spinal fever, nor is the rigidity of the spine and back of the neck present. The rose-spots of typhoid fever appear over the abdomen. The crucial diagnostic symptoms, however, include the agglutination reaction in typhoid fever, and Kernig's sign in cerebro-spinal meningitis.

Influenza is not capable of presenting any confusing degree of resemblance to cerebro-spinal fever for any length of time. In its incipiency, the sudden acquisition of fever, the severe pains in the head, back and extremities, ofttimes associated with delirium, may give rise to suspicions of cerebro-spinal fever. Properly-directed inquiry will usually disclose the sequence of events and establish the nature of the case. Very confusing, if, indeed, not incapable of recognition, are those cases of influenza associated with meningitis.

The differentiation of cerebro-spinal fever and *tubercular meningitis* is at best doubtful. Cases of the latter are not uncommon at times when cerebro-spinal fever is epidemic. Tubercular meningitis does not usually appear with the suddenness observed in cerebro-spinal fever. Lumbar puncture, as proposed by Quincke, is at times an invaluable procedure, but does not always lead to positive results. The tapping occasionally fails to withdraw any fluid ; and, even when successful, the fluid not infrequently contains either no bacteria whatever, or in numbers insufficient to make the observations conclusive. In the majority of cases it may be regarded as a reliable diagnostic procedure.

The prognosis of cerebro-spinal fever should always be guarded, as it is a very dangerous disease, the mortality ranging in different epidemics from 33 to 75 per cent.

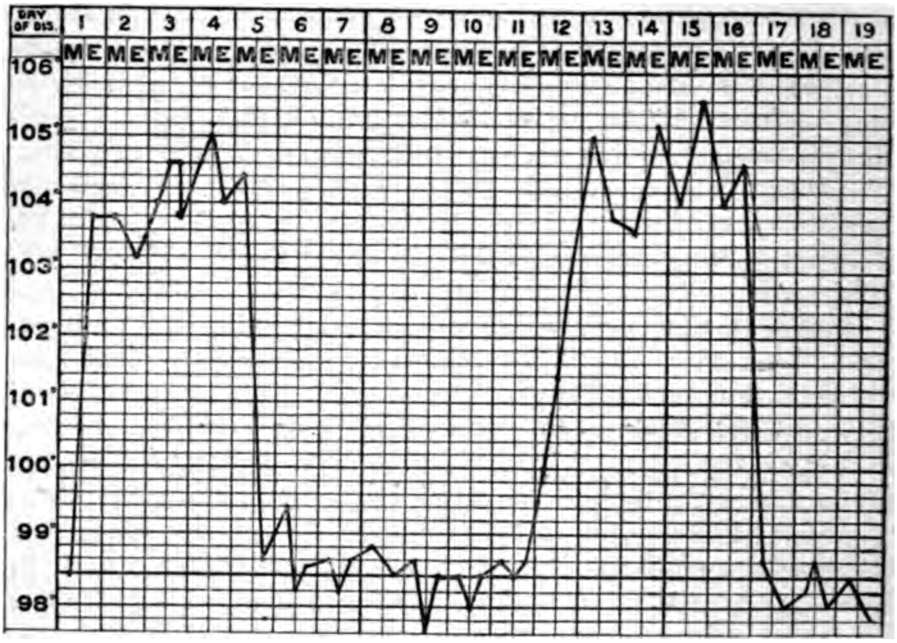
16. Relapsing Fever.

The temperature-changes of relapsing fever are characteristic and briefly described. The onset is rapid, and is associated with rigors and profound prostration. The temperature reaches anywhere from 102° to 104° F., remaining at or about this point, with morning remissions and evening exacerbations, for about a week, when it disappears by crisis. Then succeeds a period of so-called apyrexia lasting for several days, during which, however, the temperature does not always recede to the normal point. Then succeeds a fresh accession of fever, which ordinarily is not as severe as the first paroxysm. High temperature in relapsing fever does not necessarily imply an unfavorable prognosis.

This disease is observed in communities living under unfavorable hygienic surroundings, as bad air, insufficient and improper food, and filth. It occurs in epidemic form, and is dependent upon the presence of the spirillum Obermeieri in the blood during the febrile paroxysm. (*Vide* section on the Blood.)

The associated symptoms are pretty much the same as observed in the low fevers, including headache, sharp general pains, severe sweating, exhausting diarrhœa, and hæmorrhages from the gastro-intestinal and urinary tracts.

Fig. 18.



Temperature-curve of relapsing fever. (After Murchison).

The average mortality of this disease is not high. Death is usually the result of some complication, as pneumonia, abscess of the liver, thrombosis, uræmia, etc.

17. Yellow Fever.

In this disease the fever reaches its height within a few hours after the onset of the attack; certainly the maximum is not delayed later than the evening of the third day. Then the temperature begins to fall, interrupted or not by evening exacerbations. Defervescence is completed by the fifth day in favorable cases. It is succeeded by a period of depression, during which the temperature may be subnormal. Sometimes the initial fever of this disease is divided into two periods of three days each, separated by a marked remission. The temperature of yellow fever affords a valuable prognostic sign. Cases in which the maximum does not exceed 103° F. almost invariably recover. Those in which the temperature ranges from

103° to 105° F. offer a grave prognosis, the gravity being proportionate to the height of the fever. A temperature above 105° F. is strongly suggestive of a fatal issue. The fever sometimes attains a height of 108° F.

While, then, yellow fever adopts a definite type, the diagnosis of the disease is a difficult if not an impossible matter, unless it is known that the disease is prevalent in the community. Even then, an attack of bilious remittent fever occurring at such times may, because of its symptomatic resemblance to yellow fever, be incorrectly diagnosed. In bilious remittent fever the vomited matters contain bile instead of blood; the temperature-charts in the two diseases present different types of curves; the malarial parasite will be found in the blood in bilious remittent fever.

The positive symptoms associated with the fever include the slowing of the pulse with the rise of the fever, the yellowish discoloration of the conjunctivæ and skin, and albuminuria.

In the beginning of the disease the pulse is rapid in proportion to the height of the fever. After the second or third day this parallelism between temperature and pulse is disturbed, the fever continuing to increase, while the pulse drops in frequency.

Albuminuria is always present. It is first noticed about the third day. In severe cases it may appear earlier. As a rule it is transient, but in many instances it is associated with all the characteristic features of acute nephritis, the patient dying from uræmia.

The face of the patient presents some important symptoms. In the beginning it is flushed, and is associated with injection of the eyes, and tumefaction of the eyelids and lips. Careful inspection will furthermore discover a yellowish discoloration of the conjunctivæ and the skin of the face. In severe and prolonged cases of yellow fever, this jaundiced hue is a prominent feature.

In the early stage of yellow fever gastric irritability may be present, but the vomited matters consist of mucus, or possibly of a grayish fluid. In the last stage, on the other hand, there is vomiting of altered blood, giving rise to the well-known and often-described "black-vomit." This is always to be regarded as an unfavorable symptom, though not a necessarily fatal one. Hæmorrhages may take place into the skin and mucous membranes generally.

Ideas concerning the etiology of yellow fever have undergone radical changes in the past few years. This disease is now recognized as non-communicable by contact with patients or by fomites. Careful experimentation, frequently repeated, has demonstrated this beyond all question. The transmission of yellow fever is possible only through one variety of mosquito, namely, the *Culex fasciata.* Mosquitoes cannot infect themselves with the yellow-fever germ except by biting patients within the first four days of the disease. Mosquitoes themselves do not become capable of transmitting the disease until twelve or thirteen days later. Thereafter, they are ever a source of danger to whoever they bite.

18. Influenza; La Grippe.

The variety of types which cases of influenza may assume makes the symptomatology of the disease a difficult subject to epitomize. Still there are certain phenomena which are common to most cases. These include sudden onset, with fever in association with severe headache, general aching, and prostration disproportionate to the other symptoms. But even these symptoms are not identical in all cases. The type of fever in influenza is not a fixed one. It may follow a continuous, remittent or intermittent course, or it may be of a sudden or gradual onset. Ordinarily the temperature rises rapidly, and within a few hours may reach 104° F. When there are no complications, the acme is reached on the initial day of the disease, or, at the latest, the end of the second day. Generally the fever is of short duration, disappearing, as a rule, within three or four days, but in advance of the associated symptoms. Goodhart makes the statement that in many cases it ran twelve or more days, and "in not a few it ran the twenty-one-day fever of typhoid very close." It is generally believed that these persistently high temperatures are to be explained by the existence of some complications, usually respiratory. That these are not always discovered is due to careless examinations or lack of skill. The hyperpyrexia sometimes observed during the course of influenza is likewise dependent upon some complication. The possibility of influenza occurring without fever has been suggested. Analogous cases of other infectious diseases have been observed, and it is natural to believe that the same may apply to influenza.

Usually the only rigor is the one initiating the attack. Remarkable and interesting exceptions have been reported, in which the rigors were repeated daily for some time, causing strong suspicion that the cases in hand were due to suppuration.

Profuse sweating is of common occurrence after the third day of the disease. It seems to be symptomatic of the prostration.

The subjective symptoms are present from the very inception of the fever. They include pains in any and all parts of the body. The headache in particular is distressing. It sometimes drives the patient frantic,—even to delirium. The pains throughout the body are generally referred to the deeper structures, and are aggravated by any motion.

According to the type of the disease, the remaining symptoms are respiratory, gastro-enteric, or nervous. Those presenting respiratory and gastro-intestinal symptoms are generally classed as of the *catarrhal variety*. The respiratory symptoms include profuse coryza, conjunctival injection with lachrymation, harassing paroxysmal cough with or without expectoration. Physical examination of the chest discovers the signs of inflammation of the larger bronchial tubes. The cough is inclined to persist for a number of days after the patient is otherwise well. In the *gastro-intestinal* cases there are nausea and vomiting, epigastric and abdominal pain, and diarrhœa. The *nervous variety* presents the catarrhal symptoms, and, in addition, there is an exceptionally severe headache; the special senses are hyperæsthetic; delirium or flightiness is common; pulse and respiration are slow, and prostration is great.

The possible complications and sequelæ of influenza are many. Indeed, it is safe to say that there is hardly a disease which has not had one or more examples attributed to this cause. It is doubtless true that la grippe is a serious affection, owing to its after effects; but there can be no doubt that its dire influence on the health of its victims has been overdrawn. The complication and sequelæ relate almost exclusively, in well-authenticated cases, to lesions of the lungs, heart and nervous system. Of the pulmonary difficulties, bronchitis, broncho-pneumonia, and occasionally croupous pneumonia, deserve especial mention. In most cases presenting these complications, carelessness during convalescence or persistent neglect during the early days of the disease afford the proper explanation of their advent. An attack of influenza undoubtedly intensifies any predisposition to tuberculosis that may be present.

Of the cardiac complication, myocardial weakness is most commonly observed. This may be perpetuated, and eventually the patient dies of a chronic myocardial degeneration. The heart-weakness is at times a serious problem during the course of the illness.

The post-influenzal neurasthenia constitutes the most frequently observed of the sequelæ. The symptoms may consist of general weakness or prostration; they may be mental, the patient manifesting melancholia, mania, or hypochondriasis.

The prognosis of uncomplicated cases of influenza is favorable, most cases recovering in the course of a week, though required to observe precautions against exposure and over-exertion for several days longer. The complications may become quite a serious matter, especially broncho- and croupous pneumonia.

19. Miliary Fever.

In miliary fever, high temperature is a prominent feature from the inception of the disease. In grave cases it may rise to 104° or 105° F. It is sometimes preceded by severe and long-continued chills. It is associated with profuse sweating—hence the name "sweating sickness"—and severe epigastric distress. The eruption is manifested on the third or fourth day as small, irregular spots, which in a few hours give place to numerous vesicles. These dry in two or three days and form crusts, which drop off, leaving no scars.

Ordinarily the prognosis of miliary fever is favorable. In exceptional epidemics, for reasons not understood, the fatality may reach 80 per cent. of the cases.

20. The Septic Fevers.

The septic fevers include (*a*) Sapræmia; (*b*) Septicæmia; and (*c*) Pyæmia.

By *sapræmia* is meant a saprophytic intoxication. It is due to poisoning by the chemical products of bacteria. The presence of bacteria has nothing to do with it. Its symptoms result from the introduction of the poison, and are mild or severe according to the size of the dose. They cannot be increased excepting by the introduction of additional putrefying matter; there are no bacteria in the system to perpetuate the trouble, if the dose of poison is within the limits of recovery. Good examples of sapræmia are found after delivery or abortion, when portions of placenta or blood-clot are permitted to remain in the uterus and undergo decomposition; the presence of blood in the peritoneal cavity after surgical operation or abdominal traumatism. In order that the putrefying material can intoxicate, it is essential that it be in contact with a surface capable of rapid absorption. The uterine cavity and the peritoneum are good examples.

Van Lennep in his Surgical Lectures recognizes a sub-variety of sapræmia, which he designates "*septic intoxication.*" It is due to the absorption of poisons from foci of suppuration in large doses and rapidly. The symptoms are those of saprophytic intoxication. Examples of this condition are found in the sudden bursting of an abscess in an absorbing cavity; opening an abscess and not protecting the lymph spaces which have been divided; and the damming back of pus by too tight packing.

By *septicæmia* we mean septic *infection.* It is dependent not only upon the entrance into the system of the products of bacteria, but also upon the presence of the bacteria themselves in the circulation. Hence it is sometimes called *bacteræmia.* The symptoms are, however, dependent upon septic intoxication; but, by reason of the presence of the bacteria, the poison is capable of constant addition. The initial dose may be insufficient to produce serious manifestations, and yet, because of the continued generation of the chemical products of the bacteria within the body, the cases of this disease are nearly always attended by serious results. Unlike sapræmia, it is infectious.

Pyæmia is a septicæmia associated with abscess formation in various portions of the body.

(*a*) **Sapræmia.**—In the presence of wounds capable of giving rise to the conditions above stated, there will appear, as announcing the onset of sapræmia, a rigor followed very shortly by a rise of temperature to 103° F., or more. With this there will be headache, coated tongue, loss of appetite, vomiting, rapid pulse, and scanty urine. In the more severe cases, the complexus of symptoms which we are accustomed to designate "typhoid" are present. These include delirium, dry, brown tongue, sordes on mouth and lips, diarrhœa, jaundice and petechiæ. In fatal cases it is not unusual for the temperature to fall towards the end, the pulse at the same time growing weaker and more rapid. Death usually takes place on the second or third day, or it may be prolonged for a week. Favorable cases make a rapid recovery on removal of the exciting cause.

(*b*) **Septicæmia.**—Septicæmia occurs for the most part as the result of infection by the micro-organisms of suppuration, namely, the staphylococcus pyogenes aureus and the streptococcus pyogenes. Some few cases arise from the gonococcus, the pneumococcus, and the bacterium coli commune. "Although the micro-organisms do not go freely in the circulating blood, they get into it, and are carried by it and deposited in various parts; after death it is not uncommon to find in cases of septicæmia numerous capillaries throughout the body blocked with collections of cocci, which have not yet led to abscesses. In these cases the organisms have become adherent to the endothelium of the blood-vessels; and, having succeeded in overcoming the resistance of these cells, they grow there, and form the plug which is found after death." (Cheyne.)

In most cases the pyogenic cocci can be found in the blood, but in small numbers, by careful examination.

The symptoms are preceded by a period of incubation or so-called surgical fever, followed by gradually increasing "up-hill and down dale temperature-curve." The associated symptoms include anorexia, indigestion, headache, and the so-called typhoid state. There is rarely chill; other symptoms include increasing prostration, increased rapidity and weakness of the pulse, diarrhœa which soon becomes colliquative, scanty urine going on to suppression, indifference of apathy, low delirium, stupor, coma, and death. In the case of surgical septicæmia, the appearances and extent of the wound do not suggest the gravity of the general symptoms. Thus a small infected wound, as from the puncture or cut of a dissecting-knife, is succeeded by such severe symptoms as to make the generation of the poison by bacteria within the body the only conclusion possible. In these cases there are also local changes to assist in the diagnosis. These include red lines or streaks coursing along the affected extremity from the wound, and due to lymphangitis. The lymph glands themselves may suffer and become inflamed, eventually undergoing suppuration.

(*c*) **Pyæmia.**—The febrile manifestations of pyæmia are of sudden onset, although for several days preceding there may have been prodromal symptoms. There is first a severe rigor, which is followed by fever of very high degree, and this, in turn, is succeeded by a profuse and exhausting sweat. These phenomena tend to recur irregularly. The variations of temperature from hour to hour are so remarkable that a correct conception of the febrile curve can be had only by taking the temperature of the patient every two hours. To the uninitiated, the case may well simulate the phenomena of malarial intermittent, from which it differs, however, in the marked irregularity of the return of the chills, fever and sweat. The height of fever attainable is at times remarkable, 110° F. having been reported. During the intervals, the temperature usually remains about 100° or 101° F., but it may drop to normal, or even become subnormal.

The symptoms associated with the fever vary according to the distribution of the suppurative processes and the degree of infection and intoxication. The pulse is rapid and weak, and in the late stages irregular and uncountable. Examination of the blood shows a leucocytosis, and, bacteriologically, the micro-organisms of suppuration. Dyspnœa of moderate extent is not unusual. It is the result of a variety of causes, including metastatic pulmonary abscesses, pleurisy, bronchitis, or œdema of the lungs. The urine contains a small quantity of albumin, and, in the presence of a renal abscess, blood and pus. The mind usually remains clear until late in the disease; there are hyperæsthesiæ of the special senses, and general restlessness. Jaundice, erythematous rashes and the clinical signs of abscess complete the picture of pyæmia.

It may seem to the reader that a consideration of the septic fevers is out of place in a work devoted to medical diagnosis. In the present day of antiseptic surgery it is getting to be a well-recognized fact that septic infections are exceptional in surgical cases, and they do occur with remarkable frequency as an intercurrent or terminal condition of many so-called medical disorders. The number of diseases which may thus present the phenomena of septic infection is surprisingly large, and to the list may be added still others in which there is a mixed infection. Of the strictly septic disorders of special interest to the physician are phlebitis, malignant endocarditis, septic sinus thrombosis, pylephlebitis, etc. Examples of mixed infections are found in suppurative processes with infection complicating typhoid fever, croupous pneumonia, tuberculosis and gonorrhœa.

Many chronic disorders destroy life, not by reason of the special changes belonging to them, but because of infections. This subject has been studied thoroughly by Flexner. He has demonstrated that such infections are very common in Bright's diseases, arterio-sclerosis, heart diseases, cirrhosis of the liver, and other chronic disorders. The infection seems to attack with especial frequency the serous membranes, giving rise to pleurisy, meningitis, pericarditis and peritonitis.

The diagnosis of the septic fevers should not prove to be a matter of much difficulty. The initial chill in medical cases is usually interpreted as of nervous origin, but if trouble to make a temperature-observation is taken, the temperature will be found to have risen considerably. Occurring in the course of one of the acute infectious diseases, the impress of the complicating disorder upon the temperature-curve of the primary affection should lead to proper conclusions. The confounding of pyæmia with malarial fevers, even those of the pernicious type, is inexcusable. The history of a cause capable of starting sepsis constitutes an important factor in the diagnosis, but may not always be forthcoming. Those of an obscure nature, or liable to be overlooked, include nasal suppuration, carious teeth, ulceration of the mouth, rectal disease and gonorrhœa.

The prognosis of the septic fevers is decidedly unfavorable. Cases of sapræmia in which the condition is diagnosed before the patient has received too large a dose of the poison, and the source has been promptly removed, may make rapid recoveries. The majority of cases of septicæmia and pyæmia die without surgical interference.

21. Malta Fever.

The pyrexia of Malta fever is, as a rule, of the continued type ; but a few cases present a remittent or an intermittent temperature-curve. The best authorities until recently claimed that this fever was but a modified typhoid, and there was much to be said in favor of this view. It is now known that each of these diseases is dependent upon its own specific microorganism. The temperature-curve of Malta fever, moreover, presents certain remarkable departures from that ordinarily observed in typhoid. The onset of the pyrexia is more rapid than in typhoid fever. In mild, uncomplicated cases, the temperature attains 103° to 104° F. during the first week or ten days, and then drops gradually, reaching the normal at the end of the second or third week. In the typical case the temperature rises rapidly to 105° F. or thereabouts, sinking to normal in the course of the fourth week. There ensues a period of a week or ten days in which the temperature remains at normal, or a little above that point. Then comes a return of fever, which, however, is of shorter duration than the first attack ; and this, in turn, is followed by a period of comparative apyrexia. Then a third relapse occurs, and this is less severe than the second. Thus, with relapses and intermissions, the fever in ordinary cases runs a course of one hundred days and upwards.

22. Fever of Infantile Indigestion.

If, in the case of an infant or young child, a sudden rise of temperature occurs without premonitory symptoms, and if scarlatina can be excluded from the diagnosis, and evidences of local inflammation are not forthcoming, there is every probability that gastric or gastro-intestinal disease is the *casus mali.* In case of doubt, the differentiation of this fever from scarlatina is aided by the pulse-rate. In the latter, the pulse is disproportionately rapid to the height of the fever.

The fever of infantile indigestion is commonly referred to by family practitioners as gastric fever, and in this sense the use of the term is permissible. Unfortunately, however, it has also been employed to designate the mild typhoids of infancy and childhood, but here it is a misnomer. The fever of infantile indigestion has no special features other than its sudden advent. It may be continued or remittent; of long or short duration. It is limited entirely by the causative factors, namely, a mild toxæmia arising from the absorption of the products of mal-digestion.

23. Fever in Morphia Habitues.

Levenstein is the authority for the statement that morphia habitues may suffer from an intermittent fever, the result of addiction to that drug. The temperature may rise to 104° F. Quinine has no effect in subduing it, while a dose of morphia brings prompt relief.

24. Hepatic Intermittent Fever.

Pathologically, the separate mention of a hepatic intermittent fever is hardly allowable; but clinically, the use of the term is both convenient and practical because of the well-defined symptom-complex. Almost certainly it is but one of the phases of septic fever, although Charcot attributed it to the presence of a special ferment in the bile passages, and others have held that it is of purely nervous origin. Hepatic intermittent fever is especially liable to occur in connection with catarrhal cholangitis with gall-stones. The paroxysms of fever bear a remarkable resemblance to those of malarial origin, passing through the three stages of chill, fever and sweat. They are associated with jaundice, which increases in intensity with each paroxysm of fever. Fever and jaundice may persist for a long time. Cases of three years' duration have been reported. The rigors are often intense, and during the fever the temperature may rise to 105° or 106° F. The paroxysms may be quotidian, tertian or quartan. Pains in the liver and gastric disturbances are associated with the attacks.

25. Syphilis.

The existence of syphilitic fever very often escapes observation owing to the other clinical phenomena, which are, as a rule, far more obtrusive. There exists quite a divergence of opinion as to its origin. Some syphilographers plead for an essential fever, a specific effect of the infection. Others, and these constitute the majority, assert that the fever and its intensity are dependent entirely upon the severity of the associated lesions. Rise of temperature is observed just prior to the first cutaneous outbreak, and is very apt to be renewed on the appearance of fresh manifestations. The ulceration and pustular lesions in particular are capable of producing high temperature. Sometimes the fever precedes the outbreak by many days. In such cases it is not unnatural that a diagnosis of typhoid fever is made. Fournier describes the syphilitic fever as occurring in three types: (a) Intermittent; (b) continued; (c) vague, irregular type. The fever occurring during the initial stages is known as the eruptive fever. The temperature occasionally rises to as high as 104° F., usually, however, remaining below 102° F. The evening temperature is ordinarily about one degree higher than that of the morning. Its intensity seems to be favored by general debilitated conditions and the female sex.

26. The Anæmias.

The occurrence of fever in the course of the malignant anæmias is remarkably regular, though physicians do not seem to have fully awakened to this fact. The temperature-curve has no special characteristic, unless it is irregularity. In Hodgkin's disease and pernicious anæmia the temperature may rise to as high as 106° or 107° F., especially in the later stages. The remissions are marked; indeed, they may amount to intermissions. A gradual lessening of the fever may be observed during the terminal days of these diseases. The supervention of fever upon anæmia, with enlarged glands, should not lead the observer to the conclusion that an inflammatory or septic condition is about to set in.

27. Cholera Infantum.

The temperature in cholera infantum is unusually high,—sufficiently so to suggest, at times, the possibility of the case being one of thermic fever. It is usually associated with coolness of the surface, notwithstanding the fact that the thermometer in the rectum indicates a bodily temperature of from 102° to 104° F. in average cases, and in those ending fatally as high as 107° or 108° F.

28. Acute Peritonitis.

The febrile symptoms of acute peritonitis are such as to make them valuless for either diagnostic or prognostic purposes. Some cases are characterized by high fever, and others by normal or even subnormal temperature. The incidence of perforation of a hollow organ is associated with fall of temperature, as is also the advent of hæmorrhage. A subnormal temperature running for several days is often noticed in severe and fatal cases. Sometimes just before death there is a sudden appearance of hyperpyrexia, which is explained on the theory that the heat-controlling centres are disturbed by the circulation of blood contaminated with septic matter. When associated with gangrene of the bowels, the temperature is apt to follow a high range. When peritonitis finds its origin in the pelvis, the fever is generally that of septicæmia. While the above generalizations constitute a partial guide, the clinician must expect wide departures from the principles thus laid down. Local complications and constitutional states may bring about the widest possible fluctuations.

29. Appendicitis.

The general remarks made concerning peritonitis apply with equal force to appendicitis. One cannot in either disease be guided by the intensity of the febrile process to the formation of an idea as to the gravity of the local disease. Many cases of appendicitis run their fatal course with scarcely a febrile rise. Fever, then, is not to be entertained as an essential diagnostic symptom. At the same time, its association with the characteristic pain must always be regarded as having positive diagnostic value. Also the gradual lowering of the temperature, with general improvement of other symptoms, is of favorable omen.

30. Glandular Fever.

Glandular fever is not characterized by a high special temperature-curve. The temperature, as a rule, ranges from 101° to 103° F. It is a specific infectious disease without eruption, and associated with adenitis, involving especially the anterior cervical glands. The glandular enlargement and the fever attain their maximum on about the third day of the disease. The fever does not begin to fall rapidly until the last group of glands involved begins to diminish in size. The diagnosis depends upon the exclusion of other causes known to produce acute lymphatic enlargements.

31. Thermic Fever; Sunstroke; Insolation.

Two separate conditions may develop from continued exposure to hot weather, namely, *heat-stroke* and *thermic fever*. The differentiation of these is of the highest importance, for each requires radically different forms of treatment. Sir Joseph Fayrer endeavors to make a distinction between thermic fever and insolation, the latter being much the more severe of the two conditions. Personally, I cannot see that any distinction can be drawn between the affections he thus describes other than that one is characterized by more serious and further advanced symptoms.

To all outward appearances the onset of thermic fever is sudden, the patient being stricken down with the rapidity of a blow. As a matter of fact, most of the victims will admit that for some little time preceding the seizure they have suffered from prodromal symptoms, which, however, pointed to no special disorder. In the milder cases there is rapid rise of temperature to 103° or 104° F., associated with headache, nausea, vomiting, prostration, etc. In those of more severe character the temperature rises rapidly to an inordinate height, *i.e.*, to 110° F. or more. The associated symptoms in this latter class are of most alarming character; indeed, death may take place within an hour. These symptoms include coma, delirium, dyspnœa, cardiac weakness, twitching and jactitation of muscles, and even convulsions. In cases which are to terminate favorably there is a gradual amelioration of all the symptoms, and recovery may proceed without interruption. In many instances, even in those regarded as of the mild type, convalescence is slow, fever continuing for several days.

It is not unusual for serious sequelæ to follow thermic fever. Thus the patient may complain of disturbances of sight or hearing; the ability to concentrate the mind is impaired; or there may be what may be called, for want of a better name, a permanent neurasthenic state.

It does not seem probable that exposure to heat constitutes the sole etiological factor in thermic fever. The disease is especially prone to attack persons who indulge in alcoholic liquors, and who have been exhausted by physical exertion. Persons who are thoroughly acclimated are less liable to it than are others. Those who have once been overcome seem to be predisposed to other attacks on slight exposure.

Rigors.

Clinically, the term "rigor" is used as synonymous of "chill" to designate a tremor or shivering, with sensations of coldness. The phenomena comprising it are the same in any case, differing only in severity. They include general shivering or tremor, usually with chattering of the teeth; anxious expression of face; general distress; coldness of the surface of the body to the examining hand in the majority of cases; general pallor; shrivelling of the hands and fingers; blueness of portions of the bodily surface, especially beneath the finger-nails; coldness of prominent portions of the body, *e.g.*, the tips of the ears and nose and ends of the fingers; headache, nausea, vomiting, and various pains. Notwithstanding the prevalent sensation of coldness, the thermometer generally demonstrates that the bodily temperature is increased. It is claimed that the phenomena of chill depend upon a want of correlation between the surface and bodily temperatures.

Chills or rigors occur under the following conditions:

(*a*) Physiologically, from
  i. Exposure to cold.
  ii. Sensory irritation through the skin and ears; *e.g.*, the well-known "flesh-creeps" produced by peculiar rasping sensations or sounds.
(*b*) Pathologically, in
  i. Suppuration.
  ii. Malaria.
  iii. Relapsing fever.
  iv. Variola.
  v. Croupous pneumonia.
  vi. Yellow fever.

vii. Dengue.
viii. Pulmonary tuberculosis.
ix. Pleurisy.
x. Catarrhal pneumonia.
xi. Bronchitis.
xii. Inception of acute catarrhal rhinitis.
xiii. Scarlatina.

The greatest diagnostic value of rigors lies in the certainty with which their advent suggests suppuration in the course of an illness in which septic infection is a possibility, as in typhoid fever, pulmonary tuberculosis, after operations and traumatisms, etc. In such cases the thermometer may register 106° F. or more. The most characteristic rigors are observed in malarial intermittent fever, and may last for from twenty minutes to two hours, and exceptionally for an even longer period. The malarial chill has as its great characteristic its periodicity.

The prominence of chill in the symptomatology of malarial fevers leads to many diagnostic errors, as sight is too often lost of the fact that it may occur in other chronic disorders. Mistakes are most frequently made respecting pulmonary tuberculosis, in which condition, however, the chills present no periodicity worthy of the name, while the associated physical signs point to their origin.

Occurring in the course of typhoid fever, rigors are suggestive of a suppurative complication, generally of abscess or phlebitis.

Yellow fever is almost always ushered in by chills—usually, however, so slight and short-lasting as to escape observation. Exceptionally, there is a severe rigor.

In pneumonic fever, chill is a prominent initial symptom, and, though sometimes absent, is generally regarded as an important diagnostic feature.

So-called nervous chills, though not uncommon, are diagnosed far more frequently than facts warrant. In all the cases I have seen, the objective phenomena of chill are absent, the patient complaining merely of cold and creepy sensations. When such a subjective condition is accompanied by increased temperature, the diagnosis of nervous chill is entirely out of the question. It should be borne in mind that so-called nervous chills are strictly neurotic in origin, and have no place whatever in the diagnosis of malaria.

The severity of the phenomena of "rigor" can hardly find a place in framing either diagnosis or prognosis. The important factor is that they exist or have occurred. The personal equation of the patient is predominant, for chills are ordinarily controlled by the nervous system. In support of this statement, the frequency with which convulsions are substituted for chill in children and people of a convulsive habit need only be mentioned.

Subnormal Temperature.

Subnormal temperature possesses but little diagnostic value so far as suggesting the existence of a particular pathological change is concerned. It is true that certain conditions are associated with lowered temperature, and to this extent it affords valuable information. A glance over the subjoined list of influences leading to the production of subnormal temperature shows that this symptom is to be regarded as indicating certain general conditions rather than particular pathological states.

1. In healthy persons, as the result of withdrawal of heat from the body, as from prolonged exposure to cold. Such a state of affairs is unlikely to occur excepting from exposure when intoxicated, or from prolonged exposure in cold water.
2. In diseases of which malnutrition is a prominent feature. This includes diabetes, cancer, pernicious anæmia, melancholia and convalescence from exhausting fevers, cholera Asiatica, and cholera morbus.
3. Circulatory failure, as in heart diseases with ruptured compensation and shock. It may be observed that the agencies which bring about circulatory failure in shock and collapse are especially active in persons who have been already debilitated by malnutrition.
4. Hæmorrhages, when exhausting, are attended by general coldness of the body.
5. Certain nervous conditions, as injury to the dorsal region of the cord, apoplexy for the first few hours of the attack, brain tumor, general paralysis of the insane, some cases of idiocy, tubercular meningitis and cerebral tumor.
6. Sensory nerve irritation, especially in the abdominal cavity, as in renal and gall-stone colics, intestinal obstruction, enteralgia and intestinal perforation.
7. Cutaneous conditions, as scleroderma, leprosy, myxœdema.
8. Tuberculosis seems worthy of having a separate heading, as morning subnormal associated with evening rise of temperature is observed in connection with this disease with special frequency. The subnormal temperature is probably due to the combined action of malnutrition, circulatory weakness and toxæmia.
9. Certain toxæmias, including those generated within the body and those introduced from without. The former include renal toxæmia and acetonæmia of diabetic coma ; the latter, poisoning by phosphorus, atropia, morphia, carbolic acid and alcohol.
10. Heat exhaustion.

Sweat.

Properly speaking, symptoms relating to the function of the sudoriferous glands belong to the cutaneous system. The general practitioner, however, is interested in this class of symptoms, especially as related to fevers and general conditions ; hence their consideration at this time.

The sweating function may be studied as altered in one of the following three directions :

1. Increase, which we call *hyperidrosis.*
2. Decrease, which we call *anidrosis.*
3. Alterations in odor and color, known as *bromidrosis* and *chromidrosis* respectively.

Alterations in composition also occur, but have not received any practical clinical study. Thus, there is recognized bloody sweat, known as hæmatohydrosis. In certain toxæmia, as uræmia, the sweat is known to contain substances not belonging to it in health ; in jaundice, bile is an important constituent, as shown by the readiness with which sweat stains the patient's linen a characteristic yellow.

1. **Hyperidrosis.**—This may be either general or local. General hyperidrosis is an idiosyncrasy of some individuals, who sweat profusely on the slightest exertion, mental or physical, and on exposure to slight elevations of the atmospheric temperature. In the course of certain fevers it may occur as a critical phenomenon, as in intermittent fever and croupous pneumonia. Profuse sweating is not uncommon in the course of fevers, and is a prominent feature of acute inflammatory rheumatism. Occurring at the end of convalescence from acute and long-lasting illnesses, it is the result of exhaustion. In certain cases progressing rapidly towards a fatal issue, with unconsciousness, œdema of the lungs, and other evidences of approaching dissolution, sweating is sometimes profuse.

The orifices of the sweat-glands may become obstructed, leading thus to the retention of their secretion. Small vesicles, which we call sudamina, result. The contents may be clear, in which case the condition is known as *miliaria crystallina ;* opalescent, and is known as *miliaria alba ;* or the parts may be inflamed, when we call it *miliaria rubra.*

Local hyperidrosis is by no means uncommon, and is one of the minor ills of life causing great annoyance to its victims. It is apt to occur especially in the axillæ, and on the palms of the hands and soles of the feet. It is very often an expression of a lowered condition of health, neurotic or otherwise, but may occur in persons whom we regard as perfectly sound.

2. **Anidrosis.**—Like hyperidrosis, this may be a normal condition with some individuals. No matter how hot the weather, or how great the exertion they may undergo, such persons perspire but little, if at all. This

natural anidrosis can hardly be regarded as a satisfactory condition from a health standpoint, for sweating is nature's method of regulating bodily temperature when overheated from any cause. Again, the sweat constitutes an important agent for the elimination of excrementitious substances from the system. This being the case, it naturally follows that extra work must be thrown upon other excretory organs in persons who sweat but sparingly.

Anidrosis is a symptom of several common diseases. It is very prominent in *diabetes*, because of the very free elimination of water by urination, leaving little or none to be excreted by other channels ; in *Bright's disease*, and in many fevers.

3. **Altered condition** of the sweat includes offensive sweating, known as bromidrosis, and colored sweating, or chromidrosis.

Bromidrosis.—Profuse sweating, having a sour odor, is common in acute *inflammatory rheumatism*. In *renal toxæmia* the sweat may present a peculiar odor, which has been described as ammoniacal. Offensive sweating is not infrequently observed about the feet, the axillæ and the genitals. In many instances it is, of course, the result of lack of cleanliness. There are persons, however, who, despite the greatest care, cannot keep their unfortunate peculiarity from appealing to the olfactories of persons with whom they associate.

Chromidrosis is observed as a local condition, generally symmetrically distributed. It is observed with especial frequency in the axillæ. But little is known concerning it beyond the fact that it is particularly apt to occur in neurotic subjects.

In **hæmatohidrosis**, blood is found in sweat exuding from an intact skin. As most of the cases thus far reported have occurred in hysterical women, their investigation calls for most painstaking care to avoid deception. This phenomenon has sometimes been referred to as a variety of vicarious menstruation.

CHAPTER III.

THE PULSE.

From the very beginning of time, when the attention of man was directed to the alleviation of the pangs of illness, alterations in the character of the pulse were regarded as affording valuable evidence in the diagnosis and treatment of disease. It cannot be said, of course, that the theories prevailing in those ancient times had much in the way of truth to commend them to the medical men of the present day. It is interesting to note, however, that the fathers of medicine differentiated pulse characteristics, assigning to each variety some important significance. The theories employed to explain the phenomena were necessarily absurd. How could they be otherwise, in view of the fact that the circulation of the blood had not then been discovered. It was formerly believed that the arteries were full of air ; that their pulsatile movements in different portions of the body occurred independently of the action of the heart and of each other. Afterwards, when it was discovered that the pulsations of the heart and arteries were synchronous, it was contended that the dilating movements of these structures drew air into the body. Until the time of Galen, it was believed that the aorta opened into the trachea ; a belief—I might almost call it a superstition—that well shows the imperfect methods of observation of those times.

With the advanced knowledge of the present day, the indications afforded by the pulse have lost none of their importance. Indeed, increased clinical accuracy is observed here, as in all other departments of medicine. Opinions and facts are, however, stated with much less arrogance and dogmatism ; but this is to be regarded as a good sign, for dogmatism is prejudicial to the advancement of truth. Our thorough knowledge of the physiology of the circulation and the pathological conditions possible therein make symptoms of the pulse second to none in diagnostic and prognostic importance.

In the examination of the pulse, we make use of touch and the sphygmograph. The vessel usually selected as the place of observation is practically always one of the radial arteries. In convulsive patients the temporal arteries, and in infants the fontanelles, may give more accurate information. All available knowledge can be obtained only by resorting to both methods of examination. The fact that the educated touch is capable of giving all necessary information excepting in rare instances has led to the neglect of the sphygmograph by the majority of physicians. Unfortunately, it may well be said that the full value of touch is not thoroughly appreciated, for many physicians limit their investigations of the pulse to an estimate of its frequency, and occasionally to its strength, without thought of other equally valuable characteristics. A complete examination of the pulse demands attention to the following points:

1. Its frequency.
2. Its regularity.
3. Its force or strength.
4. Its tension.
5. The thickness and size of the arterial walls.
6. The condition of the arterial walls.
7. The character of onset and subsidence of the individual beats.

The most accurate results in the examination of the pulse by palpation are to be obtained only when three fingers are employed in palpating the artery. As to which hand should be employed for this purpose seems to be a matter of indifference with most physicians. I prefer to follow the advice of Broadbent, who advocates the use of the hand which will bring the index finger towards the circulatory centre. I have fancied,—but it may be only a fancy,—that the qualities of the pulse can be discerned better thereby.

1. **The Frequency of the Pulse.**—The frequency of the pulse seems like a very simple matter for observation, and yet it is often attended by erroneous results. This was well illustrated at an alleged hypnotic seance, when physicians stationed at the heart and at each wrist reported results remarkable mainly for their discrepancies.

A knowledge of the physiological factors which may disturb the pulse-frequency teaches that care must be observed in selecting a time for examination when the patient is at mental and physical rest as far as such a desirable state can be attained. The physician should not, therefore, make his investigation until such time after his entrance into the sick-room as may be necessary for the patient to regain his accustomed composure. It is also important that he count a sufficient number of beats to assure him that he has observed its average frequency. A count for a full minute, or of from seventy to one hundred beats, will generally be found requisite.

The average frequency of the normal adult pulse is seventy-two beats per minute. This normal standard may be departed from by reason of the following physiological factors:

a. Age.
b. Sex.
c. Posture.
d. Exercise.
e. Excitement.
f. Temperature.

g. Diet.
h. Time of day.

(*a*) **Age.**—The average frequency of the pulse at birth is about 140 per minute; at one year of age it is 120; at two years, about 108; at ten years, about 90. The normal standard of seventy-two is reached between the ages of fifteen and twenty years. With advancing years the pulse-rate is increased slightly, so that in the aged it may be 80 or thereabouts. Sometimes, however, the pulse of the aged is somewhat decreased in frequency.

(*b*) **Sex.**—The pulse-rate in the two sexes continues to be the same until the age of puberty, when the development of sexual and nervous peculiarities in the female results in a slightly greater frequency, which continues until old age.

(*c*) **Posture.**—The pulse is more rapid standing than sitting, and sitting than when lying down. It is generally assumed that standing increases the pulse-rate over the sitting posture by about ten beats per minute, and that lying down diminishes it by about the same number.

(*d*) and (*e*) **Exercise and Excitement.**—Under the stimulus of violent exercise and intense mental excitement, the pulse-rate may increase to almost any figure. A return to the normal should ensue very promptly after physical and mental rest. A persistence of rapid pulse after judicious exercise should always be regarded as evidence of impaired health from some cause. In the absence of any suggestions as to a possible cause, the integrity of the myocardium should be questioned.

(*f*) **Temperature.**—Intense heat is capable of greatly increasing the pulse-frequency, even to the extent of doubling it. Cold exerts a reverse effect.

(*g*) **Diet.**—The pulse-frequency is increased slightly after a meal—*i.e.*, by about five beats per minute. Animal food is said to be more active than vegetable in producing this effect. Coffee, alcohol and tea are capable of increasing the pulse-rate to a moderate extent.

(*h*) **Time of Day.**—The pulse-frequency is known to vary with the time of day, being greatest in the afternoon and evening and lowest in the early morning hours.

Increased Pulse Frequency.—The pathological causes are:

a. Fevers.
b. Asthenia
c. Pain.
d. Reflex irritation.
e. Pulmonary diseases.
f. Alcoholic excesses.
g. Excessive use of tobacco.
h. Convulsions.
i. Irritable heart.

j. Tachycardia.

k. Organic heart diseases.

(*a*) **Fevers.**—As regards frequency of the pulse in fevers, there may be formulated the general rule that for each degree of rise in temperature there is a corresponding increase of 8 to 10 pulse-beats per minute. Notable exceptions to this may be observed in clinical work; for example, typhoid fever, which is characterized in its earliest days by a rise of temperature disproportionately high to the pulse-rate; scarlatina, in which disease the pulse-frequency is disproportionately high as compared with the temperature. When, in the course of an acute febrile disorder, the pulse-temperature ratio is disturbed, it is safe to assume that more than one cause is operating to increase the pulse-frequency. What these additional causes are, is to be determined by thorough examination of the patient's illness.

A disturbance of the usual pulse-temperature relation in febrile disorders offers valuable prognostic suggestions. Thus, in the beginning of a puerperal septicæmia a rapid pulse, especially if not dependent upon high fever, is to be regarded as an unfavorable sign. Indeed, it may be that an extremely rapid pulse may be the first manifestation of the toxæmia. In all septic fevers, asthenia is associated with the rise of temperature as a factor in producing rapid pulse.

(*b*) **Asthenia.**—The pulse-rate is more or less rapid in all cases of asthenia; hence undue frequency is observed in the weakness during the course of or following all exhausting diseases. As the general condition of the patient improves, the pulse-rate approaches the normal standard. It is well to observe, in this connection, that in a given case of acute illness the rapid pulse may not be due to the same causes in the several stages of the disease. Thus, in the acute febrile disorders in their early stages, rapid pulse is one of the phenomena of fever. Still later, it may be dependent upon profound toxæmia and prostration. During convalescence, it is to be regarded as evidence of asthenia, or of cardio-vascular disease.

(*c*) **Pain.**—Pain *per se* is sufficient to cause increased pulse-frequency. I have several times utilized this fact to test the genuineness of alleged pain or sensitiveness on which claims for substantial damages have been made before a judge and jury. Given a patient claiming sensitiveness over certain spots, pressure thereon—providing the pain is genuine, and not assumed—should increase the pulse-rate. So, too, the existence of pain in the course of disease increases the pulse-rate over and above the frequency resulting from other factors of that same disease. Much depends, of course, upon the temperament of the patient, the changes being more marked in those of an emotional type, and but moderately manifested in the phlegmatic.

(*d*) **Reflex Irritation.**—Rapid pulse from reflex irritation nearly always finds its origin in gastric disorder, especially flatulent distention of

of what will eventually be a fatal cardiac disorder. Careful physical examination will discover in many of the cases some degree of dilatation of the heart; in still others we may discover central nerve lesions; while in many we are forced to the conclusion that the symptom is an expression of nervous instability similar in character producing such paroxysmal nervous diseases as epilepsy. Unlike epilepsy, it is found only in adult life.

The so-called *reflex tachycardias* originate in gastro-intestinal irritation. Careful observation will show, in these, that a long-continued auto-intoxication has produced changes in the vascular system, making the rapid action of the heart possible. The majority of the cases are engrafted upon a gouty or lithæmic basis.

Paroxysms of tachycardia are, as a rule, of sudden onset, following or not upon an apparent exciting cause. The pulse-rate rises to anywhere from 160 to 200 per minute. In one extreme case under my care it was 240. The associated symptoms are not as severe as the undue cardiac rapidity would lead us to expect. There is, it is true, some anxiety and præcordial distress, and slightly accelerated respiration. Auscultation shows, as one would expect *a priori*, that the cardiac contractions are insufficient; indeed, they seem like flutterings or vibrations. In the extreme case to which I alluded above, the patient referred to her paroxysms as "the flutters." The attacks are of extremely varied duration. In some instances they are over in five minutes, while in others they may continue for days. In long-standing cases, in which dilatation of the heart has ensued, there will be associated evidences of cardiac insufficiency, as cyanosis, pulmonary congestion, enlargement of the liver, etc.

Temporary tachycardia is always the result of some temporarily-acting or easily-removable cause.

(*k*) **Organic Heart Disease.**—In all organic heart affections, with the exception of some cases of aortic stenosis and fatty degeneration, the pulse-rate is increased with ruptured compensation. The heart endeavors to make up by frequency of action what it lacks in functional vigor.

Bradycardia; Brachycardia; Araiocardia; Oligocardia.—By the term "bradycardia" is meant a slow heart. It is generally assumed that a slow pulse necessarily proves a slowly-acting heart. This is not so, for alternate beats of the heart may not be sufficiently strong to transmit their pulsations to the wrists, and hence are not perceived in that locality. Broadbent, in his valuable monograph, lays great stress on this subject, to the almost entire exclusion of other causes of slow pulse. It is important, therefore, in every case of slow pulse, that the beats of the heart itself are also counted. As to how slow the action of the heart should be before deserving the name of bradycardia, opinions differ. It is probably well to apply the term only to cases in which the beats are less than 60 per minute. It may occur in persons who are apparently healthy in all other respects. It is a general opinion, however, that such persons are not long-lived. Under such circumstances bradycardia is said to be physiological, and is not infrequently an hereditary or family trait. It is very apt to occur in persons of tall stature and small thorax. It is said to occur more frequently in men than in women.

Like tachycardia, bradycardia may be temporary, paroxysmal or permanent. Permanent bradycardia is usually the result of organic disease. The pathological conditions in which slow pulse is symptomatic include the following:

a. Hypochondriasis.
b. Myxœdema.
c. Meningitis; brain diseases.
d. Spinal cord diseases and injuries.
e. Nephritis; uræmia.
f. Cardiac diseases.
g. Post-diphtheritic paralysis.
h. During convalescence from acute diseases.
i. Following epileptic attacks.
j. Jaundice.
k. Abdominal affections.
l. Poisons.
m. Essential bradycardia.
n. Stokes-Adams syndrome.

(*a*) **Hypochondriasis.**—The slow pulse of this condition is but part of the general sluggishness of function usually observed in such patients. The bradycardia is rarely if ever of an extreme type, 50 beats per minute probably representing the minimum frequency.

(*b*) **Myxœdema.**—Here, again, the slow pulse is part of the general functional sluggishness. It is interesting to note, in this connection, that myxœdema presents a symptomatology in many respects the direct opposite of exophthalmic goitre, the pulse in the latter disease being exceedingly rapid.

(*c*) **Meningitis; Brain Diseases.**—The number of brain diseases in which the pulse may be inordinately slow are many; especially is the symptom noted in cases attended by stupor. Worthy of special mention are hydrocephalus, tumor, abscess, paralytic dementia, hæmorrhage, and melancholia.

(*d*) **Diseases and Injuries of the Spinal Cord.**—These affect the bringing about of slowness of the pulse only when the lesion is situated opposite the fifth and sixth cervical vertebræ, although there are some who contend that injury of the seventh cervical vertebra may have a like result. The slowness of the pulse in these cases is often extreme, a rate of 20 per minute having been observed.

(*e*) **Nephritis; Uræmia.**—The slow pulse of nephritis is an expression of the toxic state, and may be regarded as suggestive of an oncoming or already existing uræmia.

(*f*) **Cardiac Diseases.**—The relation of slow pulse to disease of the heart is an exceedingly interesting question. It is well-known that in all valvular affections with the exception of aortic stenosis, rapid action is the rule. In aortic stenosis the bradycardia is the direct result of mechanical obstruction. Even in this anomaly, it is not a constant feature.

The important pathological factor in the production of slow pulse in heart disease is now believed to be obstruction of the coronary arteries. This may take place suddenly, as in embolism, or gradually. The explanation assigned for this effect of coronary occlusion is the production of irritation of the terminal filaments of the vagus in the heart. At one time slow pulse was regarded as a very important sign of degenerative changes, especially fatty, of the heart-muscle. Clinical observations have demonstrated, however, that in a minority of the cases of fatty degeneration only is the pulse slow.

According to Rosenbach, pericarditis may occasionally be associated with slow pulse, due to increase of the peripheric resistance, or "from pressure of the exudation upon the inhibitory fibres at their entrance into the pericardium."

(*g*) **Post-Diphtheritic Paralysis.**—During the course of diphtheria the onset of an inordinately slow pulse is a forerunner of paralysis of the heart. Occurring during convalescence, it has not the same unfavorable significance, though even then its appearance must be regarded with considerable apprehension, for one cannot with certainty decide that it is one of the phenomena of convalescence.

(*h*) **During Convalescence from Acute Diseases.**—Slow pulse from this cause is never extreme, generally about 60 per minute, and very rarely if ever below 50.

(*i*) **Following Epileptic Attacks.**—There are no noteworthy clinical features in the bradycardia succeeding epileptic seizure. Their nature is self-evident. Some epileptics exhibit an habitually slow pulse.

(*j*) **Jaundice.**—The slow pulse associated with jaundice is due to toxæmia. It is often quite marked, being as low as 40 per minute.

(*k*) **Abdominal Affections.**—The bradycardia associated with abdominal affections is generally assigned a reflex origin. Affections of the alimentary canal are especially important etiological factors. A slow pulse is often associated with constipation, and is a marked phenomenon in cases of intestinal obstruction. It has also been observed in connection with renal- and gall-stone colics.

(*l*) **Poisons.**—Those capable of producing slow pulse include lead, opium, carbonic-acid gas, alcohol, and occasionally tea, coffee and tobacco. The latter three, however, are more likely to produce increased frequency.

(*m*) **Essential Bradycardia.**—Under this heading are included cases admitting of no explanation, the patient enjoying good health otherwise. While it is a general tendency to attach no importance to such an anomaly, nevertheless it is a matter of observation that the possessors thereof do not live to an advanced age.

(*n*) **Stokes-Adams Syndrome; Recurrent Bradycardia.**—This condition is characterized by paroxysms of slow pulse, the rate being as low as 20 to 30 per minute, and in one extreme case reported by Heberton it was but 5. With this are associated symptoms which have been variously designated as apoplectiform, syncopal or epileptiform. The patient exhibits disturbance of the nervous and respiratory functions, vertigo, faintness, and even transient unconsciousness. Pallor more or less obscured by cyanosis is not uncommonly present. Cases of Stokes-Adams disease occur for the most part in persons of rather advanced life, and whose arteries are more or less rigid and tortuous. In many instances it seems to have been caused by some acute disease, notably influenza. It may also result from any of the factors which are capable of giving rise to cardiovascular disease. The pulse-rate is reduced between the paroxysms. It is a singular fact that the same etiological factors which may occasion tachycardia may give rise to bradycardia. Why two such opposite symptomatic conditions should arise from the same causes cannot be determined in the present state of clinico-pathological knowledge.

The distinction between ordinary bradycardia and Stokes-Adams disease is found in the fact that to the permanently slowed cardiac action of the former is added paroxysms in which the pulse-rate sinks to a still lower level, and the association at such times of cerebral, respiratory and cardiac disturbance.

Slowing of the pulse is of common occurrence during the lying-in period. The pulse-rate has been observed as low as 40 (Varnier), and only exceptionally is it at the normal. The phenomenon is usually best marked from the fifth to the eighth day, and among multiparæ than primiparæ.

The possibility of a purely nervous bradycardia has been suggested as occurring paroxysmally. Additional clinical observations are needed to demonstrate the existence of this class of cases.

The symptoms associated with bradycardia will vary greatly according to the causes. Thus, in the alleged neurotic cases (admittedly rare), there are associated dyspnœa, palpitation, and sense of constriction about the chest. These symptoms will be of sudden or gradual onset, according to the characteristics of the case.

When gastro-intestinal irritation or abdominal affections with pain are the cause, we have nausea, vomiting, severe pain, etc., to explain the phenomenon.

When the coronary arteries are diseased, we may have paroxysms of pain simulating those of angina pectoris at times, while at all times the evidences of weakened myocardium may be found on palpation and auscultation.

2. **Regularity of the Pulse.**—The normal pulse is regular in force and rhythm to a degree of nicety sufficient to excite admiration of the mechanism by which it is preserved. At the same time, it must not be forgotten that departures from this regularity may take place in response to very slight disturbing causes. Indeed, these abnormal conditions may be so slight as to give no evidence of their existence other than in their effect on the pulse-rhythm. In the absence of definite information explaining these cases, we are forced to the conclusion for the present that such arrhythmia is physiological (?), and therefore of no serious significance. Especially is this irregularity observed in infancy and childhood. If the observer is very critical in his comparisons of individual pulsations with each other, slight differences will be observed. Such exact clinical study of the pulse cannot, however, be regarded as of much value, because no information bearing on diagnosis, prognosis or therapeutics is thereby obtained. The innocence of many cases of arrhythmia must be ever borne in mind, for many a patient has had his life rendered miserable by the knowledge of the existence of irregular cardiac action. The importance of the arrhythmia in any given case can only be determined by the associated symptoms and conditions. If, for example, an irregular pulse becomes regular during the course of a fever or in response to increased muscular activity, it must be regarded as functional and of no serious import; while, on the other hand, an increase of cardiac irregularity following exertion or excitement must have the opposite significance.

Irregularity of the pulse must be studied as related to the force and the rhythm. In case of irregularity as to force, individual pulsations are observed to vary as to their amplitude; in disturbances of rhythm some pulsations are longer or shorter than are others, or there may be periods, sometimes recurring regularly, at which a pulse-beat is dropped. The term *intermittent pulse* is used to designate the latter class of cases. "Irregular pulse" covers all cases characterized by departures from the normal rhythm. An irregularity in force, like intermittency, may take place at regular intervals,—*i.e.*, every second, third or fourth beat may be weaker than its associates. Careful examination generally discovers that the intermittent pulse displays irregularities in force, inasmuch as the pulsation immediately succeeding the intermission is rather stronger than the others.

According to the character of the irregularity in arrhythmia, various types of pulse have been described. The *pulsus alternans* is one in which weak and strong beats alternate.

The *pulsus bigeminus* is characterized by alternately quick and slow intervals between the individual pulsations.

The *pulsus trigeminus* exhibits a longer interval at the end of every third beat.

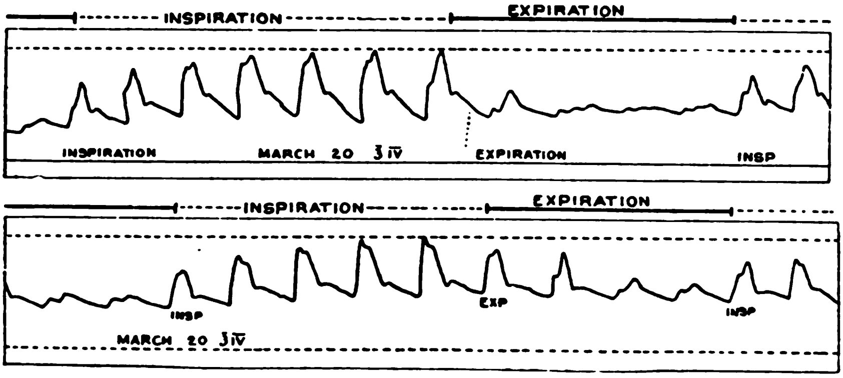
Sometimes two pulsations of unequal force are noted to occur quick together, and are followed by an abnormal pause. This is called the *pulsus bigeminus alternans.*

When the irregularity of the pulse follows a definite system,—*i.e.*, intermits or is irregular at every third or fourth beat, it is known as *allorhythmia.*

When the heart's action is irregular and tumultuous, we have the condition described as *delirium cordis.*

The *pulsus paradoxus* is characterized by weakness of the beats during inspiration. It is due to interference with the propulsion of the blood by the heart by reason of the altered intra-thoracic pressure.

FIG. 20.



Pulse-tracing of right radial in Hay's case of reversed pulsus paradoxus.

A reversed type of the pulsus paradoxus has been described by Hay, of London, as occurring in a case of aneurysm of the aortic arch. Sphygmographic tracings revealed a marked diminution of the volume of the pulse with expiration. This was explained post-mortem by the discovery that the posterior portion of the aorta in the region of the transverse arch had distended, the result being that the innominate and left carotid arteries, instead of arising from the convexity of the arch, as is usually the case, sprang from its anterior surface; and consequently, as the sternum and bony thoracic walls descended with expiration, these two arteries were caught and compressed between the thoracic wall in front and the aneurysm behind.

"*Linked beats*" are exhibited by evidence of secondary ventricular contractions in the downstroke of the sphygmogram. They are due to a repeated ventricular systole immediately following the closure of the aortic valves. This condition has been observed with especial frequency in persons convalescing from pneumonia or bronchitis.

Associated with slow pulse, it sometimes occurs in epileptics, and is then due to faulty nervous mechanism.

It is now in order to review the conditions under which arrhythmia may occur. False interpretations of this symptom in the past have been due to an altogether too narrow view of the mechanism by which the regularity of the heart's action is maintained. Lesions of the brain affecting the accelerator or pneumogastric nerves, affections of the heart itself, various toxæmiæ, and reflex irritation from the abdominal viscera, are all important factors in the production of arrhythmia. It is irritation of the accelerator and pneumogastric nerves that explains the irregular pulse associated with meningitis, neurasthenia, hysteria, and tumors of the neck and mediastinum. Irregular pulse is not infrequently observed in connection with disturbances of the auditory nerve or its nucleus. The explanation is found in the contiguity of the nuclei of these nerves, and in the proximity of their roots. The arrhythmia associated with functional nervous disturbances and shock is probably due to irritation transmitted from nerve-tissue adjacent to the nucleus of the vagus. In certain cases of arterio-sclerosis, the symptom under consideration may be present as the result of nutritional changes arising from the pathological condition of the capillaries nourishing the nuclear cells.

Among affections of the heart, arrhythmia appears to be observed with especial frequency as a result of myocardial changes, although it is sometimes a symptom in peri- and endocarditis, mitral disease, and sclerosis of the coronary arteries. Great importance has been attached to it as suggestive of myocarditis ; and yet extensive disease of the heart-muscle may take place and the pulse maintain perfect regularity. On the other hand, it should be remembered that the ganglia in the heart constitute an important part of the rhythm-maintaining mechanism, and that the delicacy of their function necessitates good nutrition, and this is secured by free anastomoses of the surrounding blood-vessels. Myocardial degeneration presupposes local malnutrition, and in all probability also disturbances of the cardiac ganglia. Hence it is right to expect irregularity of pulse when this condition exists, or when there is sclerosis of the coronary arteries.

Of the valvular lesions, those pertaining to the mitral orifice bear an important clinical relation to arrhythmia. In the case of mitral regurgitation, the irregularity is the result of the disturbance of the blood-current by the varying pressure on the thin-walled left auricle during the acts of respiration. Mitral stenosis does not seem to produce irregularity of the pulse until compensation fails and muscular changes ensue, although inequalities are not infrequent as the result of contraction of the left ventricle before its cavity is fully distended. Notwithstanding the predominance of mitral disease as a cause of arrhythmia, the possibility of other lesions producing this symptom must ever be borne in mind. Thus the *pulsus bigeminus alternans* has been observed in connection with aortic stenosis.

The reflex causes of irregular pulse are closely related to disturbance of cardiac function. Gastro-intestinal disease seems to be especially important in this connection. Sometimes a mere indigestion is the sole factor; in other cases, the pressure of a great accumulation of flatus produces marked irregularity, and even collapse and syncope. Cases of the latter class are especially frequent in neurological practice, and ofttimes require most painstaking investigation before their truly functional or neurotic character is discovered.

The stomach is not the only abdominal organ capable of acting reflexly on the heart. Arrhythmia is not uncommon during the course of recovery from surgical operations on the uterus and ovaries. It may also result from the severe pain excited by the passage of renal and biliary calculi.

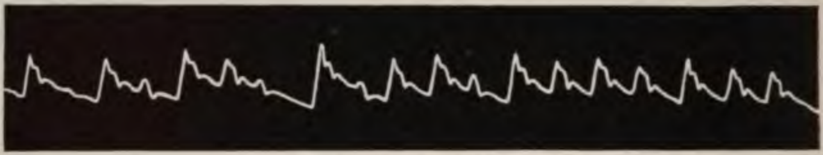
Several of the habits may, by reason of their toxic action, bring about irregular heart-action. Notable among these is tobacco. It is important to bear in mind that when arrhythmia has once been excited by this poison, and has disappeared under abstinence, it is very apt to recur on resumption of the weed even in great moderation. The irregularity produced by excessive alcoholic indulgence has more complicated etiological relations. Sometimes the toxic action of the stuff itself is an all-sufficient cause. In others, this symptom does not result until tissue-changes and exhaustion have been produced. In still others it appears only when a gouty tendency has lighted into activity.

Among other toxic causes may be noted certain of the infectious diseases (notably typhoid fever, influenza and croupous pneumonia), muscarine, tea, coffee, digitalis, aconite and belladonna.

As a rule, patients who are the subjects of arrhythmia are not aware of their trouble until informed of it by the physician. Sometimes the irregularity is manifested associated with a disagreeable sensation or discomfort in the præcordium. When the pause in cardiac action is at all prolonged, it is not unusual for it to be accompanied by an intense anxiety.

It will now be seen that arrhythmia is capable of many interpretations, according to the associated conditions. It is not by any means the serious symptom it is credited with being. Many a person has lived for years, notwithstanding its presence. It does not appear, even, that its subjects are necessarily less able to resist the ravages of acute disease than their more fortunate fellows who are blessed with regular cardiac action—providing, of course, there is neither dilatation nor myocardial degeneration. Arrhythmia appearing during convalescence from acute diseases suggests perverted nutrition of the heart-muscle, and calls for a strictly enforced rest. Irregularity of the pulse which disappears during the course of a fever, or which is suspended under mental excitement or muscular exertion, is never of serious omen. On the other hand, if the arrhythmia is intensified by slight exertion, it is to be regarded as evidence of failing cardiac power.

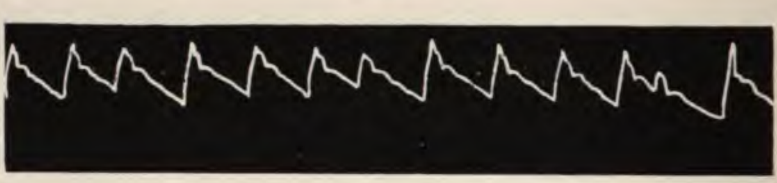
FIG. 21.



Arrhythmia showing linked beats.

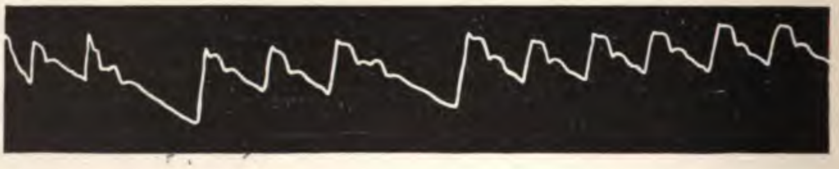
The experiments of Hering, of Prague, seem to demonstrate that careful analysis of the irregularities in the pulsus bigeminus and trigeminus affords considerable information as to the origin of the trouble. Resistance to the emptying of the ventricle excites the latter to premature contraction, the ventricles not being excited in the normal way from the auricles. The author's final conclusion is as follows: "While, on stimulation of the ventricle, the time-value of the bigeminus is equal to the time-value of two regular cardial beats, such is not the case on stimulation of the venæ cavæ and on stimulation of the auricles, excepting under certain conditions. It is therefore possible, up to a certain point, to determine, from the time-value of a pulsus bigeminus, which part of the heart was first excited by the pathological cause. These irregularities are of a myogenic nature, like the

FIG. 22.



Irregular arrhythmia.

FIG. 23.



Dropped beat; chronic myocarditis. Broad summit with well-marked dicrotism.

entire normal activity of the heart. A bigeminus of neurogenic origin is unknown."

3. **Tension.**—When the arterial tension is normal the vessel is readily compressed by the examining fingers, and empties itself between the beats. Under pathological conditions it may be increased or diminished. We speak of a pulse as of high tension when it is full between the beats. The examining fingers, rolling the artery beneath them, discover a sensation as of a cord. The first impression is that the pulsations are weak, but the stronger the compression exerted upon the vessel, the more evident do the pulsations become. This is an important point to note, for it happens not infrequently, in the absence of tests for determining the strength of the arterial impulse under different degrees of pressure, that the tense pulse may give a false impression of weakness.

High arterial tension results from the following general causes :

a. Increased volume of blood.

b. Frequent and powerful action of the heart.

c. Arteriole and capillary contraction.

Under the first of these headings we note that the pulse-tension rises after very full meals. Under ordinary circumstances the quantity of food taken is such as to cause but a temporary increase in the volume of the blood, and the influence of this is counteracted by the simultaneous relaxation of the abdominal blood-vessels. When, however, the quantity of food taken is unusually large, the tension is increased. In case of nephritis, in which the urinary water is not eliminated, there is increased volume of blood, and, of course, resulting high tension. We often note increased tension in plethora.

The ability of increased and powerful cardiac action to heighten intravascular tension is too evident to require any remarks thereon.

We find in arterio-capillary resistance the most efficient cause in the production of high arterial tension. Under the influence of external causes the vessels contract, and high tension results. This fact is of considerable importance in the etiology of apoplexy. It has long been observed that this condition is especially liable to occur during the cold months of the year. The subjects of vascular degeneration, exposing themselves suddenly to the influence of cold, bring on high arterial tension, causing rupture of the weakened walls of some one or another of the cerebral vessels. The occurrence of so-called cramps which come on while swimming, and which have been the cause of many accidental drownings, is believed to be due to increased arterial tension, excited by the vascular contraction brought about by the sudden immersion of the subject in cold water.

High arterial tension is a pretty constant phenomenon in most cases of *angina pectoris;* indeed it is held that the attacks of this disease are caused by the increased resistance which the heart meets in propelling the blood through the contracted peripheral vascular system. Practical use of this fact is made by the employment of glonoin and nitrite of amyl as palliatives. The results obtained from these remedies show the theory to be a good one, for they are generally used with excellent results by the majority of physicians.

Rigors occurring in any disease, hysteria, meningitis, migraine, and neuralgic pains generally, are all conditions in which high arterial tension may be observed.

The occurrence of high tension as the result of capillary resistance may be set down as due, in a general way, to the action of poisons circulating in the blood acting upon the capillaries. These poisons, as a rule, result from nitrogenous waste. This explains the occurrence of high tension in the course of kidney disease and gout.

High tension from arterio-capillary resistance occurs in old age, as the result of hereditary tendencies, in renal disease, gout, diabetes, lead-poisoning, pregnancy, anæmia and emphysema. The existence of high vascular tension in families is a well-attested fact. It affords the only rational explanation of the tendency of apoplexy to run in certain families, as it does also of the family tendency to sudden death from heart disease.

Renal diseases may be attended by high tension; but it is especially in interstitial nephritis that this condition is found. So important is this fact that thorough and repeated examinations of the urine are called for in all cases of high-pulse tension. It is said that when the pulse tension is low in cases of renal disease, the prognosis is unusually bad.

The high-pulse tension of pregnancy may be due to either one of two causes, namely, the presence of effete substances in the blood or increase in the volume of that fluid. In emphysema, it is due to imperfect aëration of the blood.

Next we come to consider the causes leading up to high tension. We find these in the character of the food and drink and habits of the individual. It seems that immoderate indulgence in highly nitrogenized foods, as meats, in connection with too little exercise and the taking of limited quantities of water, are important etiological factors. This suggests that in the management of these cases we should encourage the patient to limit the quantity of nitrogenous food, to drink liberally of a pure water, and to exercise freely in the open air. We also forbid the patient alcoholic liquors of all kinds, because they are causes of high tension. Especially harmful are port, sherry and beer.

We may note that constipation exerts a deleterious influence on intravascular pressure. The relief following upon a free evacuation of the bowels is often so great as to furnish an important object-lesson concerning the value of regularity in going to stool.

High arterial tension has baneful effects. Some of these are mechanical, *e.g.*, the constantly increased pressure ultimately leads to rupture of blood-vessels, an accident that is very liable to occur, because arterial degeneration is usually an associated condition. Increased work is thrown upon the heart in overcoming the capillary resistance. At first that organ may undergo hypertrophy. Later, valvular disease appears, with, finally, dilatation of the heart. The constant pressure of the contained volume of blood leads to imperfect nutrition of the arterial walls by obliterating the calibre of the vasa vasorum. Thus degenerative changes of the arterial walls take place.

Low Arterial Tension.—It is now in order to study diminished arterial tension. This condition results, speaking in a general way, from exactly the opposite causes of increased tension, namely, weakness of cardiac action, dilatation of the capillaries and arterioles, and diminution in the quantity of blood. Speaking more specifically, we find low arterial tension to result from heredity, obesity, exposure to the relaxing influence of heat, anxiety and worry, debility, food, and pyrexia. It is important to take cognizance of the family peculiarities relating to the pulse-tension, for otherwise it is an easy matter to assign a pathological interpretation to what is, so far as the individual under observation is concerned, a perfectly physiological phenomenon.

The low arterial tension of obesity can hardly be regarded as physiological. It is usually associated with weak action of the heart, and this, in turn, favors a still further deposition of fat.

Exposure to heat in any way brings about diminished arterial pressure. Especially is this true when the heat is combined with moisture. Thus, warm moist applications and exposure to a damp warm atmosphere bring about vascular relaxation. Advantage of this fact is sometimes taken by the administration of a warm bath for the reduction of vascular tension.

The influence of nervous causes in lowering arterial tension is entirely underestimated. At the same time, it must be remembered that low tension and the causes which give rise to it may of themselves cause the circulatory anomaly under consideration. The relation of cause and effect in all of these cases can only be determined by a study of the symptoms in their clinical relations. Especially are those mental conditions attended by depression important factors, as may be seen in many cases of melancholia, hypochondriasis, neurasthenia and hysteria. In many of these cases the circulatory condition is intensified by failure to take food of proper quality and in proper quantity.

All conditions tending to debility cause relaxations of the arterioles and capillaries. More than that, they weaken the heart-muscle and lessen the quantity of blood. We find good illustrations of this in diseases characterized by loss of fluids, as in hæmorrhagic disorders, and in affections having diarrhœa and diuresis as prominent symptoms. In diarrhœa there is often great and rapid abstraction of fluid from the capillaries. In cholera the lowered arterial tension finds a somewhat different explanation. There is, of course, an abstraction of fluid, but the capillaries are well contracted, thus lessening in great degree any effect of the reduced volume of blood. But the arterio-capillary contraction in cholera favors the retention of blood in the venous system, thus lowering the tension in the arteries.

Remarkable examples of sudden lowering of arterial tension are found in cases in which large quantities of fluid retained in the abdominal cavity are removed suddenly, as in the rapid withdrawal of urine from a distended bladder or of serum in ascites. This is a very important point to bear in mind in the management of these conditions, because the sudden lessening of the intra-abdominal pressure by the withdrawal of the ascitic fluid leads to dangerous and even to fatal collapse.

4. **Force.**—It would seem at first thought that the determination of pulse-strength or force is a comparatively easy matter,—one, indeed, requiring but little experience. It must be remembered, however, that certain other pulse characteristics so mask its apparent strength as to make the determination of pulse-strength a more complex problem than it appears to be. The factors which tend to confuse observation include mainly alterations in tension and variations in the size of the arteries.

As already intimated when speaking of high tension, a tense pulse may give an impression of weakness until considerable compression is exerted upon the artery, when it is found that the pulsations gain in force as the pressure of the examining finger is increased. On the other hand, low-tension pulse gives a false idea of strength which it does not possess.

The size of the artery must be taken into consideration, because cardiac contractions of a given strength will be perceived more distinctly the larger the artery to be examined. This fact is based upon the well-known principles of hydraulics.

5. **Condition of the Arterial Walls.**—The changes which may take place in the arterial walls as a result of disease include arterio-sclerosis and atheroma. The former, when at all well-marked, is easily recognized, by manipulation of the radial artery, as a thickness or undue hardness of the walls of the vessel. In the incipient stages of the disorder, however, one must rely mainly upon the recognition of an increased vascular tension.

The morbid changes are not confined to the radial artery; indeed, they are widespread. Thus the temporal arteries may be observed to be tortuous; the carotid arteries are seen and felt to beat in the neck with a heaving pulsation; the brachial arteries, like the temporal, are tortuous, and move laterally with each pulsation.

It is not too sweeping a statement to make that arterio-sclerosis is associated with interstitial nephritis, syphilis, or atheroma; that it does not exist as an uncomplicated condition.

Its association with interstitial nephritis is rendered easy of discovery by intelligent examination of the urine. The essential diagnostic factor is the failure on the part of the kidneys to maintain their normal standard of functional activity, as evidenced by the deficient elimination of urinary solids, especially of urea. Of course, the discovery of albumin and casts is of the highest importance; but these distinctive phenomena so often escape detection (if, indeed, they are always present) that one should rely more upon the presence of a persistent renal inadequacy.

The arterio-sclerosis of syphilis tends mainly to involve the cerebral blood-vessels, to the exclusion of those of other portions of the body. This fact makes it impossible to diagnose this condition sufficiently early to be of practical value by examination of arteries accessible to palpation. The knowledge that the patient has had syphilis and the presence of symptoms suggestive of disturbed cerebral function should be accepted as a hint to the wise.

Atheroma is readily recognized by manipulation of the radial arteries. The vessel under examination is tortuous and rolls under the finger like a hard cord. Its walls are found to be rough, due to the presence of small calcareous plates within its walls. In extreme cases the calcareous infiltration is so great that the artery gives the impression of a rigid rough-walled tube to the examining finger.

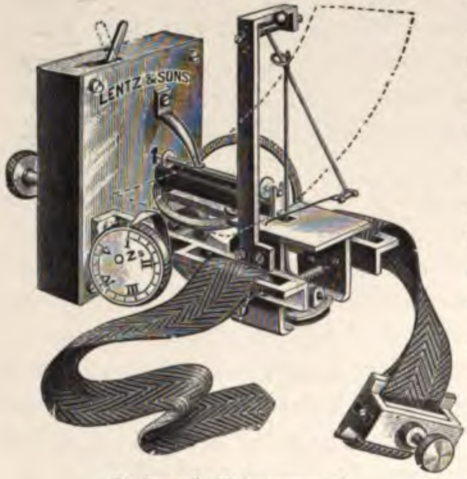
Sansom calls attention to the possibility of confounding atheromatous pulsations with the Corrigan pulse when the pulsations of the brachial artery are visible, as they often are. He directs that the examination of the brachial artery be conducted as follows: "The right arm, as well as the right side of the chest, should be uncovered; the patient should turn his face slightly to the left, and the arm should be semiflexed at the elbow. If the vessel be atheromatous, it will be seen that there is considerable side-to-side movement with each pulsation. If the arm be extended and lifted, the movement greatly diminishes. It is well known that a very great displacement of the artery, with bounding and throbbing pulsation, is seen in cases of aortic regurgitation (the *Corrigan pulse*). Then the elevation of the arm increases the suddeness and visibility. The extension and elevation of the arm serve to differentiate between the two conditions, and the diagnosis of atheroma may be confirmed by the examination of the artery by the fingers, which may detect local patches of hardness in the wall."

Unilateral Disturbances of the Pulse.—The characteristics of the pulse may not be the same on both sides of the body. Such a state of affairs may be a congenital anomaly, due entirely to anatomical differences; to pressure of morbid growths; and to vaso-motor disturbances of one limb or of one side of the body. The morbid growth most likely to produce inequality of the radial pulse is aortic aneurysm. When the ascending aorta or innominate artery is the seat of disease, the right radial pulse is weakened, or even extinct. Relative weakness of the left radial is suggestive of aneurysm of the descending aorta. Weakness and delay of the left radial may result from aneurysm of the arch between the origins of the innominate and left carotid arteries. Delay of either radial pulse may suggest various conditions on the corresponding side interfering with the arterial circulation, including pleural effusion, pneumothorax, embolism, thrombosis, tumors of the neck or axilla, and aneurysm of the subclavian, axillary or brachial arteries.

The absence of pulsations in one of the lower extremities is suggestive of arterial obstruction. The suspicion becomes a certainty when gangrene supervenes. This obstruction may result from outside pressure, as a truss, or from thrombosis or embolism.

The Indications Afforded by the Sphygmograph.—Much prejudice exists against the use of the sphygmograph in the determination of pulse characteristics. This is unfortunate, because this little instrument is capable of affording valuable information, especially when its tracings are studied in connection with the information obtained by palpation of the pulse and the physical examination of the heart. Taken by itself, it cannot, of course, give all the information desired. Indeed, what method of examination of any particular organ is capable of so doing? But there are numerous circumstances under which the sphygmographic tracing will resolve doubts as to the condition present. What more can the examiner want? Its use demands some experience and a little study, but never more than the scientific practitioner can spare.

FIG. 24.



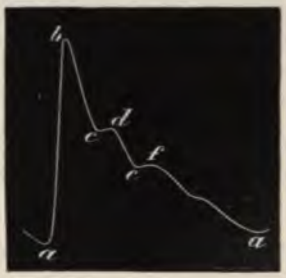
Dudgeon's Sphygmograph.

Numerous sphygmographs have been invented; but that of our own Dudgeon is the simplest, the most reliable, the most portable. It is *par excellence* the best for clinical purposes. The mechanism of the instrument can be seen at a glance at the annexed illustration (Fig. 24). The patient may be examined either lying or sitting, with the arm supported. The position of the radial artery must first be determined accurately. The button of the sphygmograph should then be placed carefully over the same, and the instrument be secured firmly in place by strapping it to the wrist. This last procedure is absolutely essential, as in this way only can an even pressure be maintained. This advice should be unnecessary, as it is a matter of common sense; but I believe it wise to mention the subject because there are numerous men who claim an ability to make correct observations without this precaution, and yet decry the reliability of the instrument.

The sphygmograph being *in situ*, there should appear at once regular excursions of the tracing-needle. If they do not, then careful hyper-extension of the hand on the forearm or regulation of the button-pressure by the little eccentric of the instrument will cause them to appear. The novice at once asks, "How much extension, and how much pressure?" To this I reply, The position and pressure which will give the most marked oscillations of the needle.

The tracings should be taken on slips of a smooth surface-paper well blackened with lampblack. To prepare these, the slip of paper should be placed in one of the tin holders belonging to the outfit, and then held over a piece of burning camphor until evenly smoked.

FIG. 25.



Typical pulse-wave. (After Sansom.)

ab, chief ascending wave; *b*, percussion or summit wave; *d*, tidal wave; *c*, notch preceding the tidal wave; *e*, aortic or predicrotic notch; *f*, dicrotic wave; *aa*, base line.

After the tracing has been made, it can be rendered permanent by immersing it in a solution of gum dammar in equal parts of gasoline and benzine. This dries in a few seconds, and can then be filed among the physician's records of the case.

The intelligent use of the sphygmograph demands, first, that the physician understands the character of the information it is capable of giving. This may be summed up in a few words: It gives an accurate record of the variations in arterial tension. The factors which bring about the variations in tension include the volume of blood thrown into the arteries with each ventricular systole, the steadiness with which this blood is maintained in the vessels by the force of the left ventricle, the apposition of the aortic valves, the elasticity of the arteries, and the resistance of the arterioles and capillaries.

The normal sphygmogram is made up of the following parts:

(*a*) An upstroke, which may be called by this title, or by the name proposed by Sansom, "the chief ascending wave."

(*b*) The terminal angle of this upstroke, generally known as the percussion or summit wave.

(*c*) A notch varying in depth in different tracings.

(*d*) A secondary wave, known as the tidal wave.

(*e*) A notch known as the aortic or predicrotic notch.

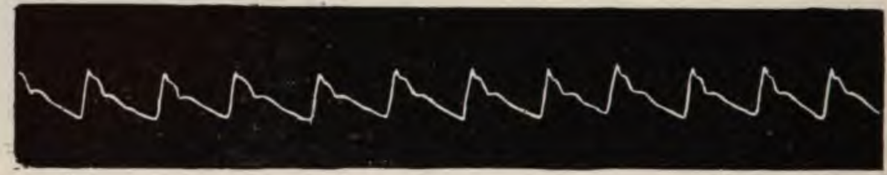
(*f*) A wave, more or less marked, called the dicrotic wave.

(*g*) The base line.

(*a*) *The Chief Ascending Wave.*—Sansom proposed the name of "chief ascending wave" for the upstroke of the sphygmogram because this title did not express any theory as to the forces giving rise to it. It is produced, of course, by the contraction of the left ventricle.

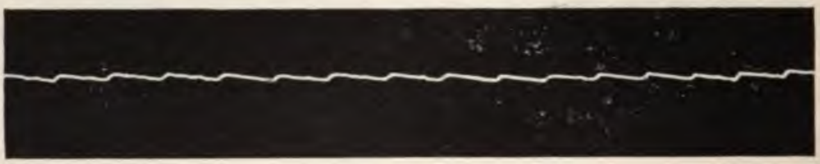
Normally, it should be perpendicular; but when the ventricle is weak, the aortic orifice narrowed, the walls of the arteries diseased, or the vascular resistance increased, it departs from the vertical, and becomes more or less slanting. At the same time the summit of the tracing becomes flattened, partly because of the retarded discharge of blood into the arteries, and partly because of the diminished momentum of the writing-needle in response to the distending force. In the case of aneurysm, the abnormal slant is observed on one side only. The aneurysm acts as an elastic bag, which, equalizing the propelling force of the left ventricle, causes the differences in arterial pressure between systole and diastole to be emphatically less evident.

FIG. 26.

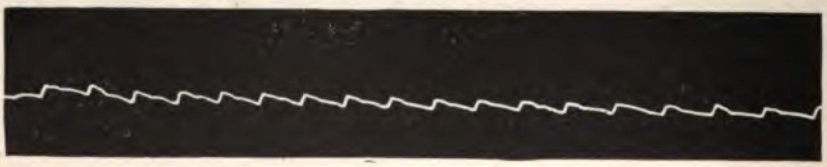


Normal tracing.

FIG. 27.



Right pulse.

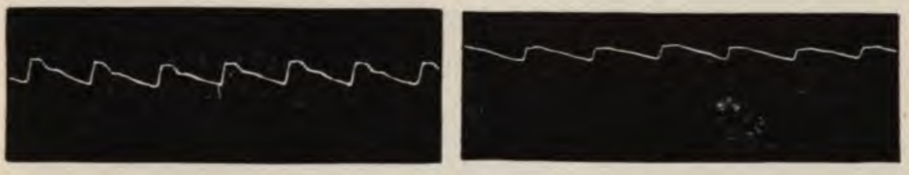


Left pulse.

Weak heart-action in inordinately stout young subject; probably extensive fatty infiltration of heart; patient liable to paroxysms of cardiac syncope; difference in two tracings due to different thickness of covering of radial arteries by soft parts.

Referring to lack of symmetry of the tracings over the two radial arteries, too much reliance must not be placed upon this symptom as evidence of aneurysm, for it may arise from other causes, or it may be the result of inexperience in the use of the sphygmograph. Thus it may be due to unilateral vaso-motor changes, as I once noted in a case of brachial neuralgia; to lack of vascular symmetry; to the deeper situation of one radial artery, etc. Asymmetry of tracings must, then, be regarded as simply suggestive of aneurysm, and not diagnostic. Of course, if one of the sphygmograms presents the characteristic features present in aneurysm, then the observation becomes of increased value. Even here, however, one must not be too sure, for cases have been observed in which the tracing on the affected presented greater amplitude than on the normal side; a phenomenon which has called forth a variety of explanations, none of which has been fully accepted.

FIG. 28.

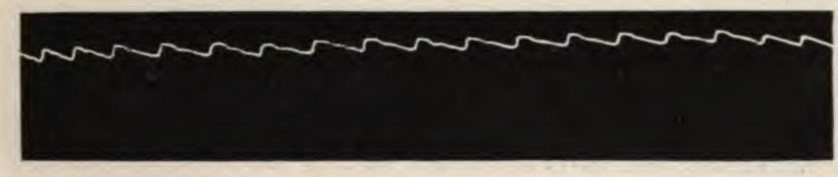


a *b*

Showing differences in pulse-tracings in a case of neuralgia of arm or malarial origin. (*a*) Tracing of the right radial pulse; (*b*) tracing of the left radial pulse (affected side).

As to the slanting of the tracing as the result of aortic stenosis, increased vascular pressure, etc., their consideration is deferred until I come to speak of the sphygmographic evidence of these conditions.

FIG. 29.



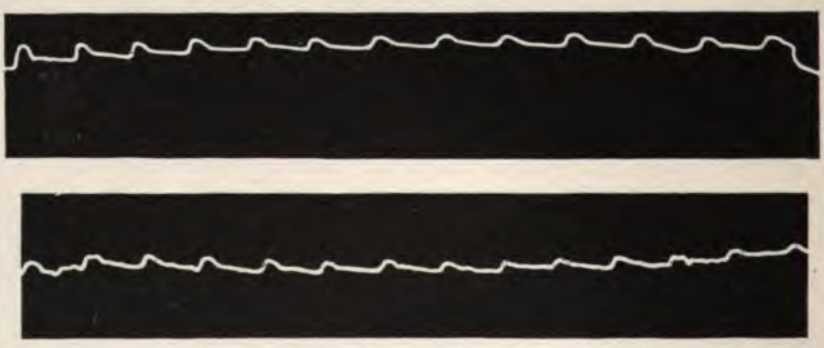
Weak pulse; myocarditis.

The chief ascending wave may slant unduly by reason of a thick layer of fat between the artery and the button of the instrument.

It would seem that the height of the chief ascending wave will vary with the force of the left ventricle. This, however, is not so, because of the variable conditions against which the ventricle may be obliged to work. Thus, with diminished arterial pressure, the line will be higher than normal; in the presence of high tension, it will be lower. When the contraction of the left ventricle is sudden, and the resistance is not increased, the recording needle will, because of the play in the levers moving it, be forced ahead at a greater speed than the distending vessel, and will attain a higher point than it would with the same degree of dilatation less energetically exerted. Height of upstroke, then, is to be regarded more as evidence of the suddenness with which the left ventricle is emptied than of strength. This statement is not an absolute one, and hence must be remembered in practice as subject to exceptions which will be suggested by the associated conditions. The quantity of blood forced into the arteries with each systole is an important factor in modifying the amplitude of the upstroke. Thus, in mitral disease, the quantity of blood being small, this portion of the tracing is correspondingly scant.

(*b*) *The Percussion or Summit Wave.*—As stated in the preceding paragraph, the play in the parts composing the levers moving the writing-needle permits the latter to be forced ahead of the dilating artery. When the needle has lost its momentum it falls by its own weight, forming an acute angle at the summit of the tracing. It continues to fall until it reaches the artery again, and then it is once more forced upwards, in the act last mentioned producing the tidal wave. In other cases the needle keeps company with the artery through the expansion of the latter, so that it descends without the recoil marking the tidal wave. Such is to be observed in the normal pulse.

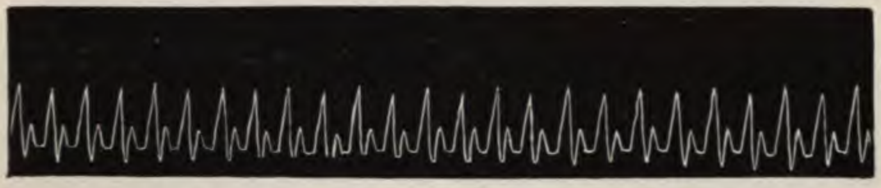
FIG. 30.



Inelastic arteries in woman, aged 35. For three years subject to chronic diarrhœa; 15 to 25 stools daily. Recovery for past year. Lower tracing taken under greater pressure than the upper. The irregularity is due to unsteadiness of the patient's hand.

(*c*) *The Tidal Wave.*—Normally the tidal wave is below the summit wave. Sometimes, however, the reverse condition obtains, and the tidal wave is higher than the summit of the percussion upstroke, and becomes rounded. We call this the *anacrotic* pulse. There may or may not be a notch between the percussion and tidal waves. When a tracing exhibits anacrotic character, care should be taken that the observation is correct, for it is very easy to secure such a tracing by reason of making too much pressure upon the radial artery. It is observed in cases of increased intravascular pressure, atheromatous arteries, and aortic stenosis. To use Sansom's words, "It indicates a prolonged effort of the left ventricle against an obstruction, but it rather shows that the ventricle is equal to the increased labor. When the ventricle fails, the bulging of the tidal wave is no longer observed, the summit of the tracing becomes flat, and dicrotism, which we consider to have an unfavorable significance, may be manifested." The tidal wave is small or absent when the heart is weak, when a strong heart is acting with full peripheral circulation, and in cases of mitral or aortic incompetency.

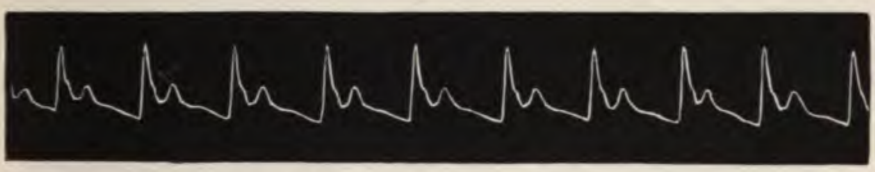
FIG. 31.



Hyperdicrotic pulse; an unusually fine example. (After Goodno.)

(*e*) and (*f*) *The Aortic Notch and Dicrotic Wave.*—These are well marked in the normal pulse-tracing, their situation being as indicated in Fig. 25. The aortic notch marks the precise moment at which closure of the aortic valves takes place, and the dicrotic wave results from the rebound of the column of blood from such closure. The diminution or obliteration of these portions of the sphygmogram are hence easily understood. Thus, in cases of aortic regurgitation, the influence of the closure of the valves is decidedly less prominent than normal, if, indeed, the notch and the subsequent wave do not disappear entirely. (*Vide infra.*) In cases of increased intravascular pressure they are almost entirely obliterated, and what remains of them occupies a much higher position in the tracing than is normally the case.

FIG. 32.



Dicrotic pulse of typhoid fever. (After Goodno.)

broad or anacrotic. Dicrotism may or may not be absent; usually, however, it is absent. The indications simulate closely the signs of high tension.

Sphygmographic Evidence of Aortic Regurgitation.—This condition, as already stated, produces the water-hammer pulse, also known as Corrigan's pulse. It is characterized by a prolonged vertical upstroke, a sharp apex, and a rapidly-falling downstroke, interrupted or not by a slight break marking the place where the aortic notch and dicrotic wave should appear.

Sphygmographic Evidence of Combined Stenosis and Regurgitation at the Aortic Orifice.—The effect of these lesions is to give a tracing in which one tends to neutralize the influence of the other. Nevertheless, when aortic regurgitation is at all well marked, the tracing exhibits, to a certain extent, the characteristic collapsing character, and the pulsations of the radial artery may be visible. Other than this, no regular rule respecting the tracings may be formulated.

Sphygmographic Evidence of Mitral Regurgitation.—Concerning this subject, we have a most excellent example of the great differences in opinion held by high authorities. Thus, Broadbent declares that this is the one valvular lesion above all others capable of producing irregularities of the cardiac rhythm, while this is strenuously denied by Balfour, Sansom and Whittaker, who assign an equal prominence in the same direction to mitral stenosis. All are united in stating that, so long as compensation is maintained, the cardiac rhythm remains regular in mitral insufficiency. Irregularity of the pulse in connection with mitral insufficiency may be said to occur only when there is co-existent pulmonary disease or failure of compensation with dilatation of the right heart. The tracings exhibit certain well-defined additional characteristics. Low tension is evident in the majority of the cases, hence the dicrotic wave is well marked. Owing to the diminished volume of blood forced into the arteries (part of the ventricular contents being forced back into the left auricle) with each systole, the amplitude of the upstroke is small. At the same time, auscultation discovers an energetically-acting heart in striking contrast with the weak pulse.

Exceptionally the tracings show increased vascular tension. Such instances may occur in connection with chronic renal disease. The sphygmograph affords valuable means of differentiating cause and effect. Thus, in the case of associated renal disease, the problem may be the determination of the influence of prolonged vascular pressure upon the mitral valves finally causing regurgitation, or the discovery of valvular disease antedating the renal complication. In the former case the tension will be high; in the latter, low. Taking, again, the case of anæmia with mitral regurgitation, the important question is the organic or functional origin of the mitral murmur. In anæmia the pulse-tension is increased, which feature is not altered by the co-existence of a functional mitral regurgitation. If the lesion is organic, the tension, on the other hand, will be low.

Sphygmographic Evidences of Mitral Stenosis.—The pulse of mitral stenosis may be either regular or irregular. In the former case it is of small amplitude, and exhibits evidence of increased tension.

In many instances irregularity may constitute an early sign of failing compensation. When not present, it may be provoked by slight muscular exertion. The irregularity may partake of almost any type; or, indeed, every possible form of arrhythmia may be observed in the same case. At the same time, it has been especially noted that secondary ventricular contractions impress their marks upon the downstroke ("linked beats").

Arterio-Sclerosis.

While, in a general way, the cause of arterio-sclerosis is advancing years,—for all sooner or later attain to the distinction of degenerate arteries,—predisposition and exciting factors play an important part in the production of the disease. The great predisposing cause is *heredity*, using this term in its broadest sense to include not only the direct transmission of a tendency to arterial degeneration from parent to offspring, but also the disposition of some families to grow old during the comparatively early years of life. It would really seem as though certain individuals were endowed at birth with limited vitality. In childhood they are adults in more ways than one, and at the early age of forty-five they are universally recognized as old men; and this premature senility is dependent upon degenerate arteries. In other instances, however, the tendency to arterio-sclerosis is more limited, and some bad habit or mode of living is necessary for its production; and here, again, certain individuals succumb to external influences much more readily than do others. Aside from heredity, the various exciting causes are made more active by certain diatheses, notably gout, rheumatism, syphilis, and the neurotic constitution. Of the exciting causes, excessive muscular exertion—a strenuous physical life—has been given a prominent place. The important factors here seem to be improperly-conducted exercise or labor, or ordinarily active exercise in persons who have not been properly trained. Numerous examples of arterio-sclerosis may be observed among machinists who have followed a life of great physical exertion. In them the arterial changes are usually secondary to cardiac hypertrophy; sometimes, however, alcohol, improper feeding and syphilis are associated exciting causes. Neurotic influences are undoubtedly productive of some cases; but here the relation of cause and effect is not always clear. Nervous activity most unquestionably produces increased arterial tension, and this, in turn, arterio-sclerosis. Thus we have an explanation of the early ageing of persons long exposed to worry, anxiety and distress. But, on the other hand, arterio-sclerosis is sometimes manifested symptomatically by nervousness, in which case the latter is an effect, and not the cause. The problem is further complicated by the influence of nervousness upon an individual's habits, forcing him into restless activity, and sometimes to habitual use of alcohol. Very important but not sufficiently recognized causes of arterial degeneration are found in certain infectious diseases and toxæmiæ, notably influenza, typhoid fever, malaria, yellow fever, gout, syphilis, and interstitial nephritis.

The symptoms exhibited by patients with arterio-sclerosis present the widest possible differences, because the clinical phenomena must vary according to the particular set of vessels which happen to be bearing the brunt of the disease. The diagnosis rests entirely upon the physical examination of the circulatory system, *i.e.*, of the heart and arteries. The radial artery at the wrist is found to present thickened and somewhat rigid walls. In some cases these changes are so far advanced as to give to the palpating finger the sensation of a tendon or quill. The tension of the pulse is increased. Changes may be found also in other arteries, notably in the temporal. This is markedly tortuous, and exhibits the same high tension. The heart is hypertrophied. In cases that have lasted for some time the apex may reach an inch or more to the left of the nipple-line. The first sound of the heart is prolonged and dull, and the second sound over the aortic area greatly accentuated. In the early stages of arterio-sclerosis, before the vascular changes become perceptible to ordinary examination methods, one is fairly safe in basing a diagnosis upon habitual high tension. Friedman and Corey have directed attention to an important early sign, namely, the lowering of the maximum audibility of the aortic sound from the third to the seventh dorsal vertebra. They explain the occurrence of this phenomenon by the alteration in the shape of the arch of the aorta by atheroma.

The most commonly observed pathological association of arterio-sclerosis is the contracted kidney; in fact, it may well be said that arterio-sclerosis cannot exist without some degree of renal degeneration. It may be that the evidence of the latter condition is not evident to ordinary urinary examinations, but careful investigations of 24 hours' specimens, with quantitative determination of urea and phosphoric acid elimination, will demonstrate the presence of permanent renal inadequacy.

Arterio-sclerosis is the fundamental factor in the etiology of many of the ailments of the syphilitic, and of persons of advanced years. As to the former cause, there is a very large group of diseases not ordinarily classed as syphilitic and yet dependent upon syphilis. The vascular system plays an important part in their production. Although the nervous system is the portion of the body most liable to suffer, many organs may be involved. The lesions are of a degenerative character, and result from lowered nutrition by reason of the diseased blood-vessels. They are not amenable to anti-syphilitic treatment because they are not syphilitic lesions.

Of the ailments of later life dependent upon arterio-sclerosis, the many nervous phenomena are of special frequency. Thus we may observe vertigo, paralyses in great variety, aphasia, convulsions, and mental deterioration.

Interference with the peripheral circulation sometimes leads to gangrene.

Sclerosis of the coronary arteries leads to angina pectoris.

The future of patients with arterio-sclerosis depends in great measure upon the care which they take of themselves. They may, for prognostic purposes, be regarded as prematurely old. When ill with some acute disorder, the outlook is rendered more unfavorable by reason of vascular degeneration ; and if obliged to undergo a surgical operation, they react but slowly. Nevertheless, careful management will do much in the way of saving life during acute illness, and retarding the advance of the arterial changes.

Phlebitis.

The causes of phlebitis include traumatism, rheumatism, gout, and other infections. *Traumatic phlebitis* may be simple or infective. The *simple variety* gives rise to little general disturbance, and finds its best examples in those cases in which it originates from clean wounds, and in which it forms part of the reparative process. *Infective traumatic phlebitis*, on the other hand, is a most serious disorder, and is attended by severe local and constitutional phenomena. The infecting material is found in the pyogenic micro-organisms, which may have been introduced at the time of the injury, or by carelessness in antiseptic technique during the operation. The character of the wound is a secondary matter, for it may have been trivial or severe ; *the introduction of septic matter is the important cause.* Such an inflammation is usually attended by a septic thrombosis. Portions of the thrombus may be detached and carried to distant portions of the body, thus setting up new septic foci. Death finally results from pyæmia or septicæmia. Sometimes the progress of the infection is prevented by the formation of an aseptic clot, thus localizing the suppurative process.

One of the most dangerous varieties of infective phlebitis is *pylephlebitis* or *portal pyæmia*, which arises from a suppuration in the area tributary to the portal vein, as in appendicitis.

Rheumatic phlebitis may occur at any stage of an attack of rheumatic fever, but is observed with especial frequency during convalescence. Occurring at this time, it is apt to be attended by a return of the articular and constitutional symptoms of the primary disease. Pain usually precedes the swelling along the course of the affected vein.

Gouty phlebitis usually involves the superficial veins. It is usually symmetrical, and assumes the fugitive or shifting character of other gouty lesions. The history of hereditary or acquired gout is usually well-defined.

The phlebitis succeeding or complicating typhoid fever is a most interesting example of an infective phlebitis. It is especially liable to occur during the stage of convalescence, and involves by preference the veins of the lower extremities. The weakness of the circulation favors the production of the phlebitis by the typhoid bacillus.

The symptoms of a simple phlebitis consist of pain, which gradually increases in severity with the progress of the lesion, and is followed in the course of a few hours or days by swelling and induration along the route of the affected vein. The parts drained by the diseased vessel become markedly œdematous if the deeper veins are involved, but not in case of disease of those superficially placed. As a rule, the swollen parts are soft; but if the obstruction is complete and in the deeper vessels, we have what has not inappropriately been called *hard œdema*. In the former case, the swelling disappears soon after the return of the circulation; the latter is apt to remain throughout life. Inflammation of the superficial veins is attended by redness of the skin over the affected vessel. The fever is usually moderate, not amounting to more than 102° F. In the typhoid cases it is preceded by a rigor.

Suppurative phlebitis presents the local and constitutional symptoms of suppuration plus those of venous obstruction. The constitutional disturbance is almost always disproportionately severe as compared with the local appearances. The fever may be of either the septicæmic or pyæmic types, characterized by marked remissions, or by repeated rigors and profuse sweats. The temperature may reach a high point—105° or 106° F. Cases of this character offer a very unfavorable prognosis, as, by this time, the occurrence of septic embolism has caused multiple foci of infection. The ordinary simple phlebitis usually offers a favorable outlook, although the possibility of embolism and its serious consequences must always be borne in mind.

Venous Pulsation.

Venous pulsation may be either physiological or pathological. As a physiological phenomenon, it is of constant occurrence in dogs and rabbits. It is occasionally observed in health in the human jugular vein. The only logical explanation then offered for its presence is that of Riegel, who attributes it to the varying velocity with which the blood flows into the right auricle. It is a purely negative pulse. The descent is made with auricular diastole, and the ascent with auricular systole.

Care is required in the investigation of venous pulsation, as, otherwise, it may readily escape detection. The patient should be in a lying position, with the neck slightly horizontal. If he is in a horizontal position, the veins will be completely distended; and if in a sitting position, the column of blood will oppose the pulsation. The phenomenon is generally most pronounced on the right side.

The physiological venous pulsation is presystolic in time. It exhibits itself as an undulation or flicker in the jugular veins at the root of the neck. It is not unusual for presystolic venous pulsation to be present in pronounced anæmia.

Pathological venous pulsation is systolic in time. It is a positive, not a negative pulse. Its ascent depending upon ventricular contraction, it can only occur when tricuspid regurgitation is present. It is, moreover, an indication of advanced lesion, because the valves of the vein must also be diseased. In the early stages of tricuspid regurgitation, it is noticeable only low in the vein. The absence of venous pulsation must not be accepted as evidence of the absence of tricuspid regurgitation, for it cannot take place when the valves of the veins are competent or the ventricles are weak.

Systolic venous pulsation in the jugular veins indicates the presence of tricuspid insufficiency, and that, too, even though the characteristic murmur of that lesion be absent. When the phenomenon is not well marked, it may be made more prominent by pressure upon the vein at about the middle of the neck.

When the pulsation of the jugulars consists of a mere undulation, it is indicative of a much shorter duration of tricuspid regurgitation.

Venous pulsation, systolic in time, occurring in the arms, may be the result either of aortic incompetence or of undue patency of the arterioles and capillaries. As is well known in aortic incompetence, the impact of the column of blood against the arterial walls is great, made so by the enormous hypertrophy of the heart and the comparative emptiness of the vessels during diastole. These great variations in blood-pressure lead ultimately to impairment of the elasticity of the arteries and dilatation of the same. Thus are favored the conditions for the production of a capillary pulse-wave, which is ultimately transmitted to the smaller veins. This venous pulsation of aortic incompetence may be observed in inflamed and erysipelatous parts and beneath the finger-nails.

In very rare instances only, jugular pulsation may be produced by a patent foramen ovale associated with mitral insufficiency. The regurgitant current passes from the left to the right auricle, and thence into the superior vena cava and jugular veins.

Venous pulsation of the jugulars may be mistaken for a communicated pulsation from the adjacent carotids. To the experienced finger, the conditions do not resemble each other. But all doubt may be resolved by making occluding pressure upon the vein midway between the mastoid and the sternum. The distal portion of the vein becomes distended. If the pulsation is communicated from the carotid artery, the distal portion will become more distended, and will pulsate stronger than before. The central portion, on the other hand, collapses, and the pulsation therein disappears.

If, however, the pulsation be venous, it will continue below the point of pressure, as before.

Pulsation of the jugular veins may be simulated by diastolic collapse of these vessels. This phenomenon occurs in certain cases with extensive pericardial adhesions, "the walls of the heart contracting with difficulty because of the adhesions, and springing suddenly into diastolic expansion, thus aspirating the venous contents." (Butler.)

CHAPTER IV.

RESPIRATION.

THE information obtained by observation of the respiration is only less in importance to that afforded by the temperature and the pulse. This fact does not seem to have been fully appreciated clinically, for it is only when the respiratory abnormality becomes decidedly obtrusive that any note is made of it. Even in hospital practice, where routine observations of the respiration are made by the nursing corps, the information given on the record-sheets is generally limited to a note of the respiratory count. It is not to be wondered at, therefore, that respiratory symptoms have been minimized in clinical importance in the minds of general practitioners.

While urging attention to detail respecting respiratory symptoms, I must utter a warning against dogmatic teaching not based on scientific foundation. Thus I can recall an observation, made years ago, to the effect that when the pulse-rate is an exact multiple of the respiration-rate, *e.g.*, 26 respirations to 104 pulse-beats, a fatal result is imminent. It is said that a lie can travel far while truth is putting on its boots, and this false teaching has been transmitted and accepted by succeeding classes of physicians ever since it was promulgated. Such a dogmatic assertion has no foundation on any principle of physiology or pathology, and must be incorrect for that reason alone, for it is with respiratory symptoms as with all others—they are interpretable on physiological and pathological principles.

The normal frequency of respiration in the healthy adult ranges from 16 to 20 per minute. The rhythm is regular. The respiratory act consists of two stages, *viz.*, inspiration and expiration, the latter being very slightly the longer.

The standard of frequency, as above stated, is subject to variations under a number of physiological conditions, as follows:

1. The frequency of respiration varies with **age.** It is more rapid in children and infants; in the latter it may be forty per minute.

2. **Exercise** exerts a great influence on the respiration-rate of all persons,—especially, however, in persons who are not accustomed to any unusual exertion,—producing increased frequency, at the same time reducing the depth of the inspirations.

3. **A rarefied atmosphere** brings about increased frequency of respiration, especially in the unacclimated. At the same time the subject

breathes more deeply, it seeming as though the increased effort was made for the purpose of getting as much oxygen as possible out of the respired air.

4. **Eating.**—There is always a slightly-increased respiration-rate after eating, especially after taking starchy food.

5. **Cold air** acts as a stimulant to the respiratory function, and increases both the number and the depth of the respirations. Hot weather has the reverse effect.

6. **Sex.**—The method of performance of the respiratory act presents marked differences in the two sexes. Adult females use the upper costal muscles mainly, in respiration, thus causing the activity of the upper portion of the thorax to be especially manifest. Adult males and children, on the other hand, use the lower costal muscles and the diaphragm, and in them breathing seems to be performed mostly by the abdominal muscles. This point is worthy of especial notice, for the presence of abdominal breathing in the female, or the upper costal in the male, is suggestive of some pathological condition. (*Vide infra.*)

7. **Sleep.**—During sleep respiratory frequency is greatly diminished; at the same time, the individual acts of respiration are less energetic than in the waking state.

While the ordinary performance of respiration is a purely involuntary or automatic act, it must be remembered that it is to a certain extent under the control of the will. Care must therefore be taken, when making observations as to its frequency or quality, that the patient's attention is not directed to what the physician is doing. Usually one can count the respirations and note their quality while ostensibly feeling the pulse. If, however, the respiratory movements are too feeble to be visible beneath the clothing, the physician can generally judge of their frequency and character by placing the hand on the chest or abdomen, as he may deem best.

In order to understand the diagnostic significance of disturbed respiration, it is necessary to have a clear understanding of **the factors necessary to the proper performance of the function.**

1. There must be furnished to the lungs an adequate supply of air of normal composition. An unduly small air-space *per capita* necessarily brings about an increased respiration-rate by way of compensation. The lessening of the quantity of oxygen in a normal amount of air has the same effect.

2. The air-passages must be sufficiently large to permit of a free ingress and egress of air. If, by reason of disease, they are unduly narrow, or if obstruction takes place, the patient breathes more rapidly than normal or makes increased respiratory efforts to compensate for the deficiency.

3. The lung tissue must be healthy and of sufficient quantity. This proposition is as self-evident as the last. If the normal quantity of air is

taken in and expired, it will not do the proper amount of work if brought in contact with a diseased pulmonary surface, or if the pulmonary-surface space is inadequate for its care.

4. The pulmonary circulation must be good. This is a corollary of the last proposition. The function of the respiration is to carry oxygen to the blood. If the lungs themselves be normal, and the normal quantity of air is respired, the respiratory function will still be imperfect unless the blood flows freely through the pulmonary vessels; for if it does not do so, it cannot be properly oxygenated.

5. The quality of the blood itself must be normal, for if it be imperfect in structure its oxygen-carrying power is diminished. Hence it is that the respiration is increased in the various forms of anæmia.

6. There must be no impediment to the free action of the lungs. That the respiratory movements may take place normally, it is essential that the clothing about the chest be loose; the muscles of the chest, the chest-walls themselves, the diaphragm and all the abdominal muscles must act freely.

In any case with disturbance of the respiratory function it is rare that any one of these causes is alone operative. The majority of cases are dependent upon the disorder of a number of the factors necessary to normal respiration.

We are now in a position to consider the

Causes Interfering with Respiration.

Their tabulation is necessarily based upon a knowledge of the physiological consideration already presented.

1. **Nervous Causes.**—Nervous influences modify the performance of every function of the human body. Those interfering with respiration are

(*a*) **Central Lesions.**—These include a common class of cases in which the respiratory centre is affected indirectly, as in all cases in which coma or stupor is a prominent factor. The respirations are increased in depth and lessened in frequency, and are often associated with a characteristic noise which we call stertor; hence the name "stertorous breathing." (*Vide* article on Coma and Stupor.) Sometimes the respiration assumes the type known as the Cheyne-Stokes. (*Vide infra.*) The intracranial lesions giving rise to the above-described state of affairs include hæmorrhage (apoplexy), embolism, thrombosis, and the terminal stages of tumors—in fact all conditions in which coma or stupor constitutes an important clinical factor. Many of these cases are associated with hemiplegia. The respiratory muscles escape involvement in the paralysis because they have bilateral representation in the cerebral cortex. (Broadbent's hypothesis.)

Lesions at or near the respiratory centre in the medulla affect the function directly, in some cases producing sudden arrest of respiration; in others acting more slowly, by reason of their chronic onset. It must be remembered that this onset-centre has most intimate connection with the cerebrum, and is readily disturbed by causes proceeding thence, as may be instanced by the influence of emotions on the respiratory act. When the centre is peculiarly vulnerable, as by disease, even death may result from fright.

(*b*) **Lesions of the Upper Portion of the Spinal Cord.**—These act because of interference with the phrenic nerve, which is a continuance of the fourth cervical nerve, reinforced by fibres from the third and fifth cervical, and by impairment of the functions of the nerves supplying the extraordinary muscles of respiration. Disease or injury of the phrenic nerves means, of course, paralysis of the diaphragm. When the lesion is in the spinal cord, as in myelitis and degenerative disease, the paralysis is bilateral; when in the phrenic nerve, unilateral. In constitutional diseases, as post-diphtheritic neuritis, both phrenic nerves may be diseased, in which case the paralysis is also bilateral.

The pathological conditions which may produce *diaphragmatic paralysis* are:

i. Spinal injuries.
ii. Acute myelitis.
iii. Acute and chronic spinal myopathies.
iv. Lead-poisoning.
v. Exposure to cold.
vi. Compression of the spinal cord.
vii. Hysteria.
viii. Infectious diseases, notably diphtheria.
ix. Disease or injury of one or both phrenic nerves.
x. Serous membrane inflammations, *e.g.*, pleurisy and peritonitis.

Paralysis of the diaphragm is to be recognized by disturbance of respiration, which becomes entirely costal. During inspiration the epigastrium recedes, instead of becoming prominent.

Phrenic or diaphragmatic neuralgia may conveniently be mentioned in this connection. It is characterized by acute pain at the lower and anterior part of the thorax, along the line of attachment of the diaphragm, and along the course of the phrenic nerves through the chest. In some cases the pain extends to the areas of distribution of other nerves, and involves the neck, lower jaw, shoulders and arms. It may be associated with angina pectoris, exophthalmic goitre, and diseases of the liver and spleen.

(*c*) **Numerous Functional Disturbances,** *e.g.*, *Hysteria.*—Hysterical disturbances of breathing are characterized mainly by the great rapidity of the respiratory acts—often as high as 80 per minute. At the same time, there does not appear to be any real dyspnœa. The pulse is rarely found to be increased in frequency. In case of hysterical laryngeal disturbance there may be actual dyspnœa. (*Vide* Larynx and Laryngeal Paralyses.)

(*d*) **Affections of the Pneumogastric and Recurrent Laryngeal Nerves.**—Respiratory disturbances arising from disease of the pneumogastric nerves are essentially the same as those originating in the respiratory centre, and are associated with symptoms referred to the various portions of the body to which fibres of these important nerves are distributed. Disease of the pulmonary branches alone is rare. Even when present, its symptomatology is not well understood, because of lack of exact knowledge of their function. It is generally believed, of course, that asthma is due to paroxysmal contraction of the muscular fibres of the bronchioles through the agency of the pneumogastric. A relation of these nerves to a form of emphysema has also been claimed. Inasmuch, however, as in all of these cases there is marked superior costal respiration, it has been suggested that the emphysema may have resulted from the energetic respiratory movements. Although the vaso-motor fibres for the lungs are derived from the sympathetic nervous system, vascular pulmonary lesions, *e.g.*, congestion and hæmorrhages, have followed section of the pneumogastric in animal and acute pontine lesions in man. Respiratory disturbance from lesions of the recurrent laryngeal nerve will be considered in the chapter on Laryngeal Paralyses.

(*e*) **Reflex influences,** *e.g.*, those derived from the stomach and skin. Sighing and yawning respirations are not infrequent attendants upon a distended or disordered stomach—sometimes a purely reflex disturbance, and at other times a mechanical phenomenon from pressure. The sudden stimulation of the skin by dashing cold water upon it causes the subject to hold his breath. The effect of cutaneous stimulation on respiration may be utilized in cases of apnœa, when the respiratory act may be started by the sudden application of cold water or by flagellation.

2. Abnormalities in the Composition of the Blood.

(*a*) **Deficient quantity** of blood, as after severe hæmorrhages, is associated with increased frequency of respiration. The rapidity with which blood is lost greatly influences the effect on the respiratory function.

(*b*) **All forms of anæmia,** *e.g.*, chlorosis, pernicious anæmia, leucocythæmia, secondary anæmia, etc. The character of the shortness of breath in anæmias closely simulates that of cardiac disease, in that it may be absent when the patient is at rest, and present only during exertion. In advanced cases of pernicious anæmia, however, it may be severe, and occur independently of any exertion. The dyspnœa of leucocythæmia and Hodgkin's disease may be brought about by a multiplicity of causes: (i) The blood-changes, as in other forms of anæmia; and (ii) by pressure of the bronchial, mediastinal and thymus glands upon the respiratory tract, recurrent laryngeal nerves, superior vena cava and its branches, the pulmonary artery, or the heart.

(*c*) **Toxæmias, including Uræmia, Fevers,** etc.—Uræmic asthma, as it is ordinarily termed, is a phenomenon of the last stage of interstitial nephritis. It is always associated with increased arterial tension and hypertrophy of the heart. Its victims are seized suddenly, in the midst of apparently normal respiration, with agonizing dyspnœa. Inspiratory efforts are violent, and the patient is obliged to sit up in order to breathe. Cardiac symptoms, as palpitation, distress and anxiety, are also important features. The seizures, as a rule, last for but a few hours, when they subside with wheezing, crepitation, and expectoration of frothy and even of bloody mucus. Attacks of minor degrees of severity may occur.

Alimentary toxæmic dyspnœa is a term including a class of cases of unknown pathology, occurring, for the most part, in gouty and asthmatic subjects. They take place mainly at night, but may be brought on during the day by exertion or excitement. They may be characterized by sense of constriction, eructations, vomiting, and cardiac excitement.

The respiratory disturbances of fevers are due to the increased temperature and the general toxæmia.

(*d*) **Deficient Aëration.**—This is noted when the respired air is deficient in quantity or quality, or both.

3. **Functional and Organic Diseases of the Heart.**—Interference with respiration as brought about by the different affections of the heart owes its existence to a variety of mechanisms, according to the particular anatomical structure at fault. In each and every case, dyspnœa in connection with heart disease means lack of compensation. It is especially liable to be the prominent feature of mitral lesions, whether stenotic or regurgitant in character. The effect of the valvular anomaly is to produce an increased blood-pressure in the pulmonary circuit, and this, in turn, produces dyspnœa, which can disappear only when compensation is established by the attainment of a proper balance between the powers of the left and right ventricles.

In tricuspid disease, dyspnœa may also be a prominent symptom. It is due to mechanical difficulty in the circulation of blood through the lungs.

In disease of the aortic valves and aorta, dyspnœa is not so prominent a feature as in the foregoing. The first evidence of ruptured compensation of aortic disease is usually found in pain and distress in the region of the heart. When dyspnœa does occur, it is due, as a rule, to local disturbance of the adjacent nerves, and to disease of the systemic arteries, which is nearly always associated. It is especially noticeable during inspiration, and is attended by marked anxiety and præcordial distress. In addition to causes above named, it may result from malnutrition of the heart-muscle, succeeding thickness or atheroma of the coronary arteries, impairment of the blood-supply of the respiratory centre in the medulla by reason of the arterial changes, and toxæmia and anæmia.

Dyspnœa is a prominent feature of all affections of the cardiac muscle; indeed, it may constitute the only symptom of a large proportion of cases. Of course it is due to impaired pulmonary circulation by reason of weakened cardiac action.

In pericarditis with effusion, the dyspnœa results from the pressure of the exudate upon adjacent nerves and other structures. Cardiac hypertrophy may give rise to the same condition, but only in very rare instances, in which the cardiac enlargement is extreme.

4. **Conditions Affecting the Apparatus Concerned in Respiration.**—These include:

(*a*) **Certain Painful Affections.**—The interference with respiration will vary according to the situation of the pain and the character of the disease giving rise to it. Thus, in pleurisy or pleurodynia, or other painful affections of the chest, the respiration will be almost entirely abdominal; in case of peritonitis or diaphragmitis, the respiration will be entirely costal. In unilateral pleurisy the thoracic movement will be restricted to one side. Pleurodynia or intercostal rheumatism will have the same effect as pleurisy, though less obtrusive.

(*b*) **Paralysis and Spasm of the Diaphragm.**—The former of these has been sufficiently elaborated when referring to interference with the nervous mechanism of respiration. We need now, therefore, refer only to diaphragmatic spasm. This may be either tonic or clonic. In tonic spasm of the diaphragm, the lower portion of the chest appears to be enlarged and the voice is weak; dyspnœa is prominent. Clonic spasm of the diaphragm, aside from hiccough (*vide infra*), occurs only in association with other convulsive phenomena, *e.g.*, epilepsy, uræmic convulsions, etc.

(*c*) **Organic Changes of the Muscular, Bony and Ligamentous Structures of the Walls of the Thorax.**—These include changes producing rigidity or softness of the thoracic walls, as malignant disease, muscular atrophy, ossification of cartilages, bony deposits, fatty degenerations, and such alterations of the structure of the diaphragm as atrophy, displacement, hernia, perforation and tumor.

5. **Obstruction of the Air-Passages.**—This may exist in any portion of the respiratory tract, from the nasal passages to one of the main bronchial tubes. While nasal obstruction interferes considerably with normal breathing, it rarely gives rise to marked breathlessness, as the patient is apt to use the mouth as an alternative air-passage. As for the throat (including under this term the fauces and pharynx), it is somewhat unusual for disease in this situation to obstruct the passage of air to such marked degree as to produce dyspnœa. Yet such a condition may arise

in acute suppurative tonsillitis and diphtheria, and still more rarely in phlegmonous, pharyngeal and tonsillar inflammation. Dyspnœa from laryngeal obstruction is comparatively common in diphtheritic laryngitis, but less so in other affections of that organ. (*Vide* Larynx.) Obstruction of the trachea is characterized by dyspnœa, mainly expiratory, but which, however, is not of so high a grade as that observed in connection with laryngeal stenosis. Depression of the soft parts about the thorax during inspiration, notably the supra-sternal fossa and intercostal spaces, takes place, but not until the obstruction is well advanced. Auscultation sometimes discovers a stridulous breathing with a focus of especial prominence, which generally marks the situation of the tracheal stenosis. In most instances of dyspnœa dependent upon tracheal disease the larynx participates also in the pathological process. Tracheal obstruction of sudden onset is practically always dependent upon the introduction of a foreign body. Dyspnœa can only result from bronchial obstruction when one of the larger tubes is involved. The condition is then characterized by absence of the vesicular murmur over the pulmonary tissue tributary to the stenosed tube, limitation of the respiratory movements on the diseased side, normal percussion resonance, and sometimes sibilant and moist *râles*. The pathological conditions which may produce tracheal and bronchial obstruction are (i) goitre, (ii) aortic aneurysm, (iii) enlargement or suppuration of the mediastinal glands, (iv) malignant disease of the œsophagus, (v) foreign bodies in the air-tubes, (vi) syphilitic and other forms of ulceration, with their resulting cicatrices. It is well to note, in this connection, that foreign bodies are more liable to lodge in the right than in the left bronchus, because the opening into the former is the larger.

6. **Physical Conditions Independent of the Respiratory Apparatus, but Interfering with it in Various Ways.**—These act mechanically, either by pressure upon the nerves controlling or directing respiration, or directly upon the thoracic viscera themselves. The primary lesion may be in either the thorax or the abdomen. In the former are to be noted the various forms of tumors, mediastinal abscess, and serous effusions. Of the abdominal lesions, tympanitic distention of the stomach and bowels, ascites, hepatic enlargements, ovarian cysts and pregnancy occur with especial frequency, and disturb respiration.

7. **Conditions of the Pleuræ.**—Diseases of the pleuræ disturb respiration in various ways: (i) By the pain excited by the respiratory act, as during the first stage of pleurisy, and (ii) by diminution of the air-space in the lungs by pressure, as in hydrothorax, empyema, the second stage of pleurisy; *i.e.*, the stage of effusion. In empyema the dyspnœa is not always by any means in direct ratio to the accumulation of pus, as the rapidity with which the process takes place and the general condition of the lungs themselves must be taken into consideration.

8. **Conditions of the Lungs.**—Pulmonary disorders disturb respiration by a variety of mechanisms: (i) By interference with the pulmonary circulation, as in embolism, in which case the dyspnœa is extreme and of sudden onset; (ii) by destruction of lung-tissue, as in advanced tuberculosis; (iii) by interference with the passage of air through the bronchial tubes, as in catarrhal inflammations, with swelling of the mucous membrane and the associated secretions; (iv) by disordered innervation, as in asthma; (v) by diminution of the breathing-surface by deposit of tubercles. Niemeyer held that shortness of breath from this cause could be excessive, notwithstanding the absence of physical signs to reveal the presence of the tubercular deposits. It is worthy of note that the dyspnœa in pulmonary disorders is influenced largely by the rapidity with which the pathological changes have taken place—as witness the great dyspnœa ensuing upon pulmonary embolism, or the introduction of a foreign body and the marked shortness of breath in pulmonic fever—and the comparatively little disturbance attendant upon destructive pulmonary lesions of gradual progess.

Next taking up the studies of the varieties of respiratory disturbance, it is convenient to continue the use of Roberts's classification as a basis. It is as follows:

1. **Deficient respiration,** which includes:
   - (*a*) Slow breathing.
   - (*b*) Restrained breathing.
   - (*c*) Shallow and feeble breathing.
   - (*d*) Ineffectual breathing.
2. **Dyspnœa,** or difficulty of breathing, which includes
   - (*a*) Obstructive or inspiratory dyspnœa.
   - (*b*) Excessive breathing, or ordinary dyspnœa.
   - (*c*) Shortness of breath.
   - (*d*) Dyspnœa, orthopnœa.
   - (*c*) Paroxysmal dyspnœa.
3. **Peculiar disorders of respiration,** including
   - (*a*) Cheyne-Stokes breathing.
   - (*b*) Hiccough.
   - (*c*) Hysterical breathing.
   - (*d*) Sighing, yawning, etc.

1. **Deficient Respiration.** (*a*) **Slow Breathing.**—This may be observed in sleep in some healthy persons, the respirations being as few as ten per minute. Pathologically, slow breathing (brachypnœa) occurs in certain nervous affections and toxæmiæ. The respirations in these cases may be of normal depth, or they may be unnaturally shallow. Slow and shallow respiration may be observed in hysteria, trance, and shock. In the cases of slow breathing from cerebral disturbance, the pathological condi-

tion brings about a state of unconsciousness also, and the respiration is noisy (stertor), as in apoplexy, opium poisoning, uræmic coma and acute alcoholism, though just as often the respirations of uræmia may be increased in frequency. The stertorous breathing of apoplectic states (using this term in a general sense) is aggravated when the patient is lying on his back, and generally brings about an increase of intravascular pressure.

Certain cases of slow breathing seem to be brought about by reflex disturbance. Strubing reports two such. In one it was dependent upon "irritation of the inspiratory fibres of the superior laryngeal nerve during acts of swallowing," and in the other it seemed to be excited reflexly by irritation of the trigeminal fibres in a case of chronic rhinitis with turbinated hypertrophy."

(*b*) **Restrained Breathing.**—This, as a rule, is dependent upon some painful or mechanical condition which restricts the movements of the chest in its entirety or over a limited area. Sometimes it is one side of the chest that is involved, so that the movements of that part lag behind the other. Ordinarily, this is evident to inspection. Occasionally it is necessary to employ palpation in order to detect it. "Lagging" over the apex, it may be well to note in this connection, sometimes occurs as a sign of early phthisis. Occurring over the lower part of one side, it is a sign of pleurisy, pneumonia, and pulmonary infarction.

The effect of pain is to cause the affected part to assume the position of expiration, while respiration is carried on by means of the abdominal muscles.

The diseases which produce restrained breathing are pleurodynia, pleurisy, and angina pectoris.

In cases of restrained breathing the respiratory act may be increased in frequency; more rarely, however, it is slowed.

(*c*) **Shallow and Feeble Breathing.**—Aside from those cases in which it occurs as part of the phenomena of an hysterical seizure, shallow and feeble breathing is to be regarded as a very unfavorable sign, occurring as it does, in the majority of cases, with impending dissolution. It permits of but little interchange of gases by respiration. It ultimately gives place to gasping breathing. It is often associated with loud, moist *râles*, audible even at a distance from the patient, and due to pulmonary œdema.

The absence of respiration is accepted as one of the signs of death. Shallow and feeble breathing may be so complete as to make it appear to the ordinary methods of examination that respiration has ceased. Under such circumstances, even feeble breathing can be discovered by placing a very highly-polished mirror over the patient's nose and mouth, when the slightest respiration will produce a foggy appearance, contrasting strongly with the brightness of the balance of the mirror's surface.

(*d*) **Ineffectual Breathing.**—Here we have a condition in which efforts to breathe produce no respiratory movements, or, at the best, but feeble ones. It occurs in conditions in which the pleural cavities are distended with fluid, *e.g.*, in hydrothorax, empyema, and pleurisy, with effusion; in distention of the air-vesicles, with loss of elasticity of the lungs, *e.g.*, emphysema, asthma, and paralysis and rigidity of the respiratory muscles.

2. **Dyspnœa.** (*a*) **Obstructive Dyspnœa; Inspiratory Dyspnœa.**—The terms "inspiratory" and "expiratory" dyspnœa are in great measure relative. It is rare, indeed, that the interference with respiration is limited to either the inspiratory or the expiratory period. In each case the act is disturbed throughout. The only purely inspiratory dyspnœa is that accompanying paralysis of the laryngeal abductors. The obstructive dyspnœa of laryngeal diphtheria, etc., is generally referred to as inspiratory, but observation shows that expiration is not unhampered. For a further exposition of this subject the reader is referred to a previous paragraph in this chapter (page 127), and to the chapter relating to laryngeal symptoms. Inspiratory dyspnœa may depend upon weakness of the chest-wall and the inspiratory muscles, as in rachitis.

(*b*) **Excessive Breathing; also called Ordinary Dyspnœa.**—The type of ordinary dyspnœa or excessive breathing is found in the breathlessness resulting from great exertion. The respirations are increased in frequency,—indeed, they may attain the rate of sixty per minute,—at the same time their depth is greatly increased. There is no pause succeeding the respiratory act. Expiration no longer depends upon the natural elasticity of the respiratory mechanism, but becomes an active muscular movement. The extent to which dyspnœa appears succeeding active muscular exercise is largely dependent upon personal factors. Those of sedentary habits get out of breath on any slight exertion, especially if, as is usually the case, they are handicapped by an undue amount of adipose. The trained athlete, on the other hand, is able to perform difficult and prolonged muscular feats without any disturbance of this character. Ordinary dyspnœa occurs pathologically when moderate work is demanded of crippled organs; also in the course of fevers, anæmias, toxæmias, pleuritic and abdominal effusions, pneumonia, bronchitis, heart affections and tuberculosis.

(*c*) **Expiratory Dyspnœa.**—Expiration is normally the weaker portion of the respiratory act, and hence is liable to greater involvement in any case of breathlessness. At the same time there are conditions in which expiration especially is embarrassed, these including diseases in which the elastic expiratory force of the chest and lungs is impaired, *e.g.*, emphysema, asthma, and rigidity of the walls of the chest.

The only pure expiratory dyspnœa arises from obstruction, and is due to a movable growth which acts as a ball-valve.

(*d*) **Orthopnœa.**—This is simply an aggravated dyspnœa, the patient demanding the sitting or upright position in order to breathe with greatest advantage. While it may occur in connection with any of the diseases capable of producing intense dyspnœa, it is observed with especial frequency in cases of advanced heart disease, acute croupous pneumonia, asthma, aneurysm, and other intra-thoracic tumors.

3. **Peculiar Disorders of Respiration.** (*a*) **Cheyne-Stokes Respiration.**—The typical Cheyne-Stokes respiration consists of cycles of a peculiar disturbance of the respiratory rhythm. At the start the respirations are both slow and shallow, and with each set increase in rapidity and depth until the maximum in force and frequency is attained. Then the respirations recede in both particulars as gradually as they increased. Then there is a pause of several seconds, followed by a recurrence of the phenomenon. As a rule, each cycle consists of about twelve to fifteen respirations, but never over thirty. The patient may be conscious throughout, or he may be conscious during the active stage and unconscious during the pause.

Atypical cases have been described. Thus a deep sighing at intervals has been described as Cheyne-Stokes respiration. Stokes found this condition in connection with gastric and hepatic troubles. Some cases cease with a sigh at the maximum of the cycle, after which there is a pause, and the phenomena are renewed.

Biot's respiration is generally regarded as a modification of the Cheyne-Stokes. It is characterized by periodical pauses in the midst of normal respiration.

Cheyne-Stokes respiration has received a variety of explanations, none of which can be regarded as at all satisfactory; hence it seems to be useless to theorize concerning it. We do know, however, that in any event it is a sign to be regarded with grave apprehension. It has been observed in the course of a variety of brain affections, including cerebral hæmorrhage, tumor, abscess, meningitis, in heart diseases with ruptured compensation, in renal toxæmia, diffuse peritonitis, typhoid fever and other acute infectious diseases, and morphia poisoning. In each of these it is a sign of most unfavorable character. It does not necessarily indicate a fatal issue, for sometimes patients having it recover. Its significance is less unfavorable in acute infections, as typhoid fever, than in brain, heart or kidney disorders. It does not always indicate that the fatal termination, though inevitable, is near at hand, for patients have lived with it for weeks. One case is reported by Sansom in which it lasted over three months. A modification of the Cheyne-Stokes respiration is occasionally noted in apparently healthy persons during sleep. Of course it then has no sinister meaning.

Biot's respiration has the same prognostic significance as that of Cheyne-Stokes.

(*b*) **Hiccoughing: Singultus.**—Hiccough is a peculiar clucking sound of respiration due to clonic spasm of the diaphragm. As a rule, it is an insignificant symptom, and of short duration. Occasionally it may prove obstinate, and, continuing for days, and even months, may be a matter for considerable alarm. In chronic diseases, especially in renal toxæmia, typhoid fever and peritonitis, it is an unfavorable prognostic sign. Hiccough may result from the following conditions:

i. Gastric disorders of all kinds.
ii. Gormandizing, especially in infants and children.
iii. Tobacco.
iv. Highly-seasoned food.
v. Peritonitis.
vi. Typhoid fever, in which disease it may be suggestive of perforation.
vii. Hysteria. It can hardly be regarded as a serious symptom in hysteria, no matter how long may be its duration. In one of my cases belonging to this category the paroxysms had continued over a period of eight months, and yet the young woman gave no evidence of ill-health in consequence.
viii. Brain disease, especially in the terminal stages.

(*c*) **Hysterical Breathing.**—Here we have a term which may be used to indicate a great variety of peculiarities of the respiratory act. They are to be recognized by their failure to conform to any of the respiratory types in their etiological relations above mentioned, and by their association with the well-known though often not easily recognized stigmata of hysteria. Rapid breathing of a peculiar type, which can be better appreciated when seen than from reading a description thereof, is a common phenomenon of hysterical paroxysms of all sorts, and in doubtful cases becomes a valuable point for differential diagnosis.

(*d*) **Yawning, Sighing,** etc.—Ordinarily, yawning occurs as an expression of fatigue. It consists of a deep inspiration, with elevation of the soft palate and uvula, and at the same time the mouth is stretched wide open, and the arms, and sometimes the legs, are fully extended,—the whole act ending with a loud expiration. Yawning is to a certain extent a nervous phenomenon, as one may frequently witness in an assemblage where, when one person starts a yawn, numerous others are unconsciously impelled to imitate him.

CHAPTER V.

THE DIGESTIVE TRACT.

The Mouth, Including the Lips, Buccal Cavity, the Gums, and the Tongue.

THE anatomical structures entering into the formation of the mouth consist of mucous membrane, connective tissue, muscles, glands, and teeth.

The pathological lesions include congestions, inflammations, eruptions, ulcerations, tumors, and caries and necrosis of the maxillary bones.

The Lips.

The clinical study of the lips demands observations as to their color, motility, shape and size, and the presence or absence of eruptions, fissures, etc.

(*a*) **Color of the Lips.**—Pallor of the lips is observed in all forms of anæmia; blueness results from general chilling of the body; cyanosed hue from heart and pulmonary diseases; and a yellowish discoloration from jaundice.

(*b*) **Movements of the Lips.**—*Tremor* of the lips occurring in the course of acute illness is significant of general asthenia. It is occasionally observed, though very rarely, in neurotic subjects under pressure of great mental emotion. It is one of the early signs of general paralysis of the insane, in which case it will probably be associated with tremor of the tongue and hands, and the characteristic mental and physical phenomena of that disease. *Twitching* or *restlessness* of the lips occurs in chorea, and is then associated with choreic movements in other portions of the body.

Paralysis of the lips is a prominent feature of glosso-labio-laryngeal paralysis (bulbar paralysis). In the early stage of the disorder it occurs as a simple weakness of the parts, associated with a similar condition of the tongue. It is then characterized by an inability to approximate the lips perfectly, in consequence of which the patient is unable to whistle or to pronounce the labial sounds, as e, u, p, b and m. When the weakness is pronounced, there is dropping of the lower lips.

Unilateral labial paralysis is due to disease or injury of the seventh cranial nerve on the corresponding side. There is imperfect closure on the affected side, with involuntary escape of saliva. There is also distortion of the face. (*Vide* Face Symptoms.) It also forms part of the facial paralysis in cases of hemiplegia.

(*c*) **Abnormalities in the size and shape of the lips** result from quite a variety of conditions. Occurring congenitally, thickness with protrusion of the lips is noted in idiots and cretins. In children it is one of the phenomena of the scrofulous constitution (scrofulous hypertrophy of the lips).

In adults it is observed normally as a racial peculiarity (as thick lips in negroes), and pathologically in myxœdema (thyroid cachexia).

Swelling of the lips, attended by pain, heat, and increased redness, indicates *cheilitis*, and is nearly always the result of traumatism or chemical action. Suppuration may result.

Gradual inflammatory swelling of the lips (*cheilitis glandularis*) is accompanied by but little pain; the lips become greatly indurated and immovable, and there is a secretion of tough, viscid mucus. As in acute cheilitis, there is a tendency to suppuration.

Inflamed and swollen lips, with marks suggesting that the parts have been bitten, may be observed after epileptic and other types of convulsions.

Thinness of the lips is due to atrophy of the muscular tissues, and occurs in glosso-labial palsy and unilateral facial paralysis. Thin lips are normal to some individuals. They occur as a racial peculiarity in Indians.

(*d*) **Eruptions,** etc.—*Hydroa*, commonly known as fever-blisters, appear on the lips in a number of acute diseases. They are particularly common in severe colds, typhoid fever, malarial fevers, and croupous pneumonia. They appear regularly on the lips of some women during menstruation.

Linear cicatrices and *fissures* on or about the lips of infants constitute important evidence of hereditary syphilis. The diagnosis is usually rendered certain by the association of mucous patches on the oral mucous membrane. The tissues about the fissures are infiltrated; hence the infantile patient exhibits a puckered appearance about the mouth. The infiltration also serves to produce eversion of the edge of the fissures. A vertical crack on the middle of the lower lip is without special diagnostic value.

In adults these fissures do not, as a rule, have any important significance, being due, for the most part, to seborrhœa, eczema, coryza, etc. They are especially apt to occur at the median line of the lips and in the commissures.

Chancre of the lips may result from infection through these fissures, generally in the act of kissing, though occasionally by drinking from contaminated cups. They are observed especially on the lower lips at the median line. They are usually single, but may be double, occupying adjacent portions of the upper and lower lips. They are nearly always circumscribed, and have an indurated base. In many instances, however, the infiltration involves the entire lips, while in still others it is not readily discerned.

All indurated ulcers of the lips are not chancre. Indeed, the most frequently-observed ulceration with induration, in this locality, is *epithelioma.* It appears usually on the lower lips as a warty tumor, gradually enlarging, and finally ulcerating. It scabs over time after time. It usually runs a course of several years, the glands beneath the jaw being finally involved, as shown by their enlargement. Epitheliomata occur, for the most part, at or after middle age.

The Mouth.

In the examination of the mouth, attention should be paid to the condition of the mucous membrane, the presence or absence of swellings, and the character of the secretions.

Investigation of the condition of the mucous membrane demands attention to its color, the presence or absence of ulcerations, deposits and false membranes.

Color of the Mucous Membrane.—*Pallor* of the mucous membrane is found in all forms of anæmia; *undue redness* is observed in all inflammations and scarlatina; a *patchy redness* in measles; a *yellowish tinge* in jaundice; a *cyanotic hue* in heart diseases with ruptured compensation; *brownish* or *bluish-black spots* in Addison's disease; *blue stains* in some cases of lead-poisoning; purple spots in purpura.

Inflammation of the mouth is known as *stomatitis.* Of this there are several varieties, as follows:

Undue redness of the mouth, with sensation of heat and soreness, without ulceration, with increased secretions, indicates *catarrhal stomatitis.* It is the result, usually, of inattention to cleanliness. The gums may become swollen and spongy from the continued irritation and inattention enforced by the inflammatory tenderness.

The formation of small, yellowish-white spots, breaking down into shallow ulcers with raised margins, indicates *aphthous stomatitis.* The individual ulcerations are referred to as *aphthæ.* They are quite painful, and interfere with mastication. They are associated with more or less inflammatory redness of the balance of the mouth. It is observed, for the most part, in children during their first dentition.

The formation of small, painful ulcerations on the mouths of nursing-women constitutes the affection known as *stomatitis materna.* It is probably a variety of aphthous stomatitis.

The inflammation begins on the free edge of the gums and the folds of the mucous membrane between these parts and the cheeks or tongue, accompanied by spongy swelling of the gums, which bleed easily; the inflammation is followed by ulcerations, irregular in outline though sharply drawn, having a yellowish base, with irregular, swollen, bluish-red edges; horrible fœtor of the mouth. The disease is *ulcerative stomatitis.*

Mercurial stomatitis conforms to the description of ulcerative stomatitis above given. Sponginess of the gums, great salivation, metallic taste in the mouth, and the ulcerations, are the important symptoms.

The inflammation starts as a dark- or bluish-red hard spot, increasing in size and deepening in color with more or less rapidity. Finally there is a general induration, and one or more blebs form. Rapid breaking-down of tissue ensues, with perforation and extensive destruction of the soft parts. Fœtor is terrible; prostration is profound; the disease generally progresses to a fatal termination. This is *gangrenous stomatitis*, or *noma.* It occurs only in greatly-depraved constitutions. It is one of the rare sequelæ of measles in the poorly nourished.

An appearance of **raised white patches,** looking very much like curd of milk superimposed upon the mucous membrane, constitutes the characteristic lesion of *thrush*, or *mycotic stomatitis.* Careful examination shows that the deposit is adherent to the mucous membrane, and, when forcibly removed, leaves an abraded surface. Its occurrence is limited, for the most part, to infants in poor general health, and to adults who are in the terminal stages of an exhausting disease, as pulmonary tuberculosis. It is dependent upon a fungous growth known as the *oidium albicans.*

White patches may also be indicative of that rare disease, *pemphigus vegetans.* The initial symptom is severe pain on eating and swallowing, after which the white patches, which are more or less detached, appear. The eruption of pemphigus appears on the cutaneous surface some days, or even weeks, later. The blebs rupture and undergo deep ulceration. The disease is a very serious one, those cases in which the eruption is at all extensive being invariably fatal.

An inflammation of the mouth may result from local disease or injury of any kind. In such cases, the inflammatory changes will be limited strictly to the parts adjacent to the seat of the primary lesions, thus pointing to the vicinity in which search should be made for the source of irritation.

The increased redness of the mouth in *scarlatina* is the mucous membrane analogue of the cutaneous eruption. It is contemporaneous with the exanthem, and is associated with a similar redness of the pharynx and fauces. The same remarks hold true respecting the patchy redness of the mouth in *measles.* In this connection it is well to observe that *variola* or *small-pox* is attended by mouth lesions starting as papules and proceeding to vesiculation, and thence to pustulation; in other words, following the exact course of the cutaneous eruption. At the same time, it must be borne in mind that the eruption must be modified by the physical conditions prevailing in the mouth, the heat and moisture leading to early disintegration of the lesions.

Of great diagnostic importance are certain spots appearing in the mouth, in cases of measles, from one to three days before the cutaneous manifestations of the disease. These lesions, now universally known as "**Koplik's spots,**" are especially present on the mucous membrane of the cheeks and lips. They do not appear on the soft or hard palate or in the fauces. They consist of small, irregular, bright-red spots, in the centre of which is a minute bluish-white speck. With these lesions fully developed and present in great numbers, the mucous membrane of the mouth presents a bright-red color, studded by the bluish-white specks. Not only will this condition enable us to diagnose measles before the appearance of the eruption, but it will also prove of great value in the differentiation of measles in the eruptive stage from other diseases which simulate it.

Of the ulcerations of the mouth, those of ulcerative and aphthous stomatitis have been described already. It remains, therefore, to speak only of those dependent upon gastric disorder, nervous influences, syphilis and tuberculosis. Persons with weak stomachs are not infrequently the subjects of small ulcerations resembling the aphthous ulcers. Individual crops are of short duration, but the tendency to recurrence is a source of great annoyance. The same remark applies to the so-called neurotic sore mouth.

The **syphilitic ulcerations** are as common as the tubercular are rare. They may appear either in the secondary or the tertiary stage of the disease. In any case, the history of the patient is an important factor in reaching a conclusion. At the same time it is necessary to pay attention to the clinical features of the ulceration, for they possess certain well-defined characteristics which, if studied in conjunction with co-existing signs and symptoms, make a diagnosis fairly positive on the objective appearances alone.

Syphilitic lesions in the mouth, as elsewhere, are remarkable for their multiformity. At the same time, it must be borne in mind that the varied objective appearances presented are dependent upon well-defined rules of pathology, the proper understanding of which is important for their ready recognition. The buccal lesions of syphilis are the mucous membrane prototypes of the cutaneous manifestations of the disease, and are especially apt to be contemporaneous with the same. Owing to the physical conditions existing in the mouth, namely, heat, moisture, great vascularity, and constant motion, these lesions are very liable to undergo characteristic changes. Pathologically, syphilitic lesions are the result of small-celled infiltration and connective-tissue proliferation.

Syphilitic buccal lesions include erythema, papules, pustules, and tubercles; and each of these may, as the result of the degenerative changes, break down to form ulcers.

The *syphilitic erythema* exhibits itself as a diffuse dark congestion having a sharply-defined border, situated generally on the soft palate at about the part where it joins the hard palate. In other cases it starts as circumscribed rounded patches which rapidly become confluent, forming patches also with well-defined borders, but with tortuous margins. Lastly, the erythema may start as a punctate eruption over the palatine vault and the mucous membrane of the cheeks and lips.

These various erythematous lesions not infrequently take on degenerative changes, and then *erosions* or *superficial ulcerations* result.

The important data for the recognition of the above-described lesions are, aside from the history of the case and the presence of similar cutaneous manifestation, the bluish-red color of the hyperæmia and their location at the junction of the hard and soft palate.

The **papular lesions** are rarely observed in their characteristic state, because the physical conditions present lead to rapid changes. They constitute one of the most frequently-observed of the buccal manifestations of syphilis, and their secondary changes lead to the formation of the well-known mucous patches.

Mucous patches, or **mucous plaques,** as they are also called, start usually as round, red spots. They may appear in any portion of the mouth, though usually in situations where the mucous membrane is subject to irritation, as at points of contact between the cheek and a ragged tooth-edge; at the point of contact with the pipe; on the tongue, where hot tobacco-smoke habitually irritates. Not infrequently they are observed on opposing mucous surfaces—not, however, by reason of auto-infection, but because of contact-irritation. They may present an oval, circular or irregular outline; they are usually raised somewhat above the surrounding mucous membrane; their surface is covered with an exudate which gives it an opaline appearance. Sometimes this deposit is so thick as to give it the appearance of a false membrane.

The membranous covering may be exfoliated, in which case there is presented a varnished-like appearance, bright or reddish-brown in color, and it, in turn, quickly becomes covered with a membranous deposit. Mucous patches are to be differentiated from non-specific lesions, with which they may be confounded by the absence of a surrounding inflammatory areola, unless there be secondary stages; in which case, such areolæ may result from irritation, as in other lesions of the mouth.

Ulcerations of syphilitic origin sometimes appear as a spontaneous lesion, so to speak, both in the hereditary and in the acquired disease. In the former, such ulcerations may be round, oval, or horseshoe-shaped. In the latter, they appear as small ulcerations upon the lips, cheeks, and fauces; sometimes the ulcerations are large, though superficial, with irregular borders, especially upon the palatine arch. Ulceration may also appear in conjunction with fissures, concerning which more will be said when treating of these last-named lesions.

The above remarks apply to what is generally known as the secondary stage of syphilis. In the tertiary stage, mouth lesions are also common. They differ from those of the secondary in their greater tendency to dryness, marked proliferation of tissue, and obstinacy to treatment. Thus the mucous patches of this period are dry, show more or less thickness, and are very resistant to treatment. Sometimes they appear as circinate or serpiginous scaly patches. These may degenerate into the condition known as leucokeratosis. This last-named lesion, appearing on the buccal mucous membrane, is especially to be located on that portion of the cheek which is in contact with the teeth when the jaws are closed. Starting as an irregularly-shaped plaque, it increases in size, by the formation and coalition of new lesions or by direct extension, until the entire inside of the cheek is involved as far back as the teeth extend. The whole surface presents a dull, soggy appearance, and is divided and subdivided into minute lines and fissures. The central portions are whiter than the periphery. The lines may radiate from a central point, *e.g.*, the commissures of the mouth or the central white points above referred to.

Reference to gummata may seem inappropriate at this particular time; but it is deemed best to study these lesions now, because of the frequency with which they form the foundations for buccal ulcerations in tertiary syphilis. *Gummatous tumors* vary greatly in size—all the way from the size of a pin-head to that of an olive, in fact. They give rise to but little inconvenience excepting from their size, and are recognized by their objective appearances. They may be circumscribed or confluent, single or multiple; and they may start in either the mucous, submucous, muscular or periosteal tissue. The overlying mucous membrane is greatly injected. Physical conditions of the mouth and the nature of the lesions tend to early and destructive ulceration; in fact, irreparable damage may be effected within a day or so after the discovery of the tumor. Occurring on the hard or soft palate, they may lead to perforation in an incredibly short space of time. Sometimes, indeed, perforation takes place before the existence of the gumma is even suspected.

False membrane on the mucous surface of the mouth may occur in association with a similar condition of the pharynx (*diphtheria*), constituting *buccal diphtheria*. When cast off, it leaves a raw surface. Microscopical examination will discover the Klebs-Loeffler bacillus. It is sometimes called *membranous stomatitis*.

An ulcero-membranous stomatitis has been described. Discrete patches of thick, yellowish pseudo-membrane or exudation appear. These are separated from subjacent tissues by an ulcerative process, leaving erosions. This condition arises as the result of bad hygienic surroundings and improper food. It is rarely seen among persons in comfortable circumstances. It is especially liable to appear in children between the ages of five and ten years during the course of convalescence from acute diseases. It begins, as a rule, with gingivitis, after which a thin, yellowish exudate appears, and may involve the pharynx and fauces. Mild cases recover in the course of a few days, while the severe ones proceed to deep ulceration. It may run a course of a month or more. Its prognosis, even in the severe cases, is favorable.

Ulcerations, known as the **sublingual ulcer,** are not infrequent in the course of whooping-cough. They occur beneath the tongue on either side of the frænum, and are believed to be the result of injury of these parts against the edges of the teeth during the violent paroxysms of coughing.

Simple ulcerations are occasionally observed in the mouths of dyspeptics. They yield readily to treatment, but exhibit a remarkable inclination to recurrence. They are sometimes known as "*canker-sores.*"

Small, superficial ulcerations on either side of the median line of the hard and soft palate, surrounded by redness of the adjacent mucous membrane, and accompanied by some pain, occurring in children, constitute the condition known as *Bednar's aphthæ.* It is not a serious affection.

Perlèche is a form of ulceration which begins usually at the angle of the mouth as a small fissure. By constant licking and irritation infection takes place, and there is formed an intractable ulcer of considerable size. It generally runs a course of two to four weeks.

Perforating ulcer of the mouth is sometimes observed in the course of locomotor ataxia. It has been ably described by Baudet and Fournier. In the beginning there is noted progressive absorption of the alveoli, with dropping out of the teeth. Then come the ulcerations, which are atonic, indolent and chronic. Later there may be perforations of the palatine vault. Fissures may form between the mouth at one end and the nasal cavities or maxillary sinuses on the other. Fournier calls the affection "*mal perforant buccæ.*"

Riga's disease is a rare affection of the mouth, due to the production at the level of the frænum of a prominent fungoid excrescence covered by a whitish exudate. It is said by some to be an anæmic affection; by others, to run in families. It is observed with especial frequency in children at the time of their first dentition. The previous state of health does not seem to be an important etiological factor, as it affects the cachectic and healthy alike, although it does not constitute a source of danger, excepting in those whose systems are greatly "run down."

Soggy, dull-gray patches, due to thickening of the epithelium, are often seen on the mucous surface of the cheeks and lips in dyspeptics, anæmic subjects, and others who are in depreciated health.

Cloudy patches, with casting-off of epithelium, is very common among inveterate smokers, and are generally described as "smokers' patches."

Tumors.—The tumors of the mouth are gummata (already referred to), ranula, salivary calculi, and various malignant neoplasms, notably epithelioma.

Ranula is a painless cystic swelling presenting a bluish, pellucid appearance, occurring on one side of the frænum linguæ. Its contents are perfectly clear. It occurs, for the most part, in adults, and gives rise to no inconvenience excepting from its size.

Salivary concretions consist of particles which are first recognized generally by the tongue, when lodged in one of the salivary ducts, usually in that of Steno. When producing more or less complete occlusion, there may be considerable inflammatory swelling of the cheek or salivary gland.

Shape of the Mouth.—Arching of the hard palate and alterations of the dental arch are observed in mouth-breathers as a result of post-nasal adenoids. It is one of the consequences of habitual thumb-sucking in infants, and is not infrequent in idiots and other degenerates.

Additional Remarks Concerning Some of the Diseases of the Mouth Not Fully Described in the Preceding Pages.

1. Catarrhal Stomatitis.

Catarrhal stomatitis is usually due to traumatism or lack of cleanliness. The traumatism is brought about by sucking at various substances, as rubber nipples, pieces of ivory, etc., and by the eruption of teeth. In adults it is most frequently produced by excessive use of tobacco and indulgence in hot or highly-seasoned foods. It may also be symptomatic in disorders of the stomach and bowels.

The symptoms include pain and heat as subjective phenomena, with redness and swelling—which may be general or local—of the buccal mucous membrane. In the initial stage there is dryness of the mouth; later, there is a free production of viscid mucus, and some slight fœtor of the breath.

The prognosis of the disease is invariably favorable, all cases recovering under proper care within a week.

2. Aphthous Stomatitis.

The prevalent view concerning the etiology of aphthous stomatitis is that it is dependent upon constitutional, and not upon local causes. It has been observed in conjunction with quite a variety of conditions, as pneumonia, the acute exanthemata and gastro-intestinal disturbances, and is especially liable to occur in infants during the teething period; hence dentition has been one of the assigned causes. It may occur in adults without apparent cause, and has been observed in women during pregnancy, lactation, and at the menstrual period.

For two or three days prior to the outbreak of the lesions the little patient may exhibit symptoms such as might characterize the onset of any acute ailment. On the other hand, the first manifestations, aside from a slight irritability, may consist of ulcerations. Next the lesions appear as small whitish or yellowish-white spots, surrounded by an areola, and occurring singly or in groups. They may extend to and involve the pharynx. In the course of twelve to thirty-six hours the epithelial layer is cast off, and there remains a small, superficial ulcer. It is usual for the aphthæ to appear in successive crops; so that, while individual lesions may continue for only a few days, the course of the disease may be quite prolonged. As a rule, there is considerable pain, arising not only from the aphthæ themselves, but also from the associated general stomatitis.

The prognosis is invariably favorable. The only serious effects from aphthous stomatitis arise from the interference with nutrition by reason of the child's refusal to take food, and infection of the ulcerations.

The only affection with which aphthous stomatitis can possibly be confounded is *foot and mouth disease*. The latter affection is associated with a high grade of stomatitis and severe constitutional depression.

3. Mycotic Stomatitis. (Thrush.)

The primary cause of this affection is the fungus oidium albicans. It is necessary, however, that there be a predisposition, and this is brought about by uncleanliness and cartarrhal stomatitis. Constitutional depreciation, as debility, cancer, tuberculosis and old age, are also important causes. Infection is generally carried by dirty nipples or feeding-bottles.

The subjective symptoms are generally mild, the disease not being recognized until the discovery of the lesions. Objectively there appear one or more white blotches, which resemble curds of milk in their appearance. They are firmly attached, owing to the fact that they consist of the fungus intimately interwoven with the buccal epithelium. When detached, a bleeding surface is left behind.

The prognosis of thrush is favorable, recovery ensuing promptly under treatment; sometimes without treatment. Occurring in the course of cancer, tuberculosis, etc., it is to be regarded simply as one of the terminal symptoms.

4. Ulcerative Stomatitis; Putrid Sore Mouth; Stomacace.

Ulcerative stomatitis occurs exclusively during the tooth-bearing ages. The majority of cases appear in children between the years of four and ten.

The etiology is found in general bad hygienic conditions and infection. The specific agent in the latter class of cases remains undetermined. Predisposition to the disease has been excited by such acute affections as scarlatina, measles, whooping-cough, pneumonia, typhoid fever, and malaria. Mercurial poisoning produces lesions indistinguishable from those of ulcerative stomatitis.

The initial changes consist of a swelling and sponginess of the gums, the swelling and inflammation spreading to the adjacent portions of the cheeks, the mucous membrane of which takes the imprint of the teeth. The gums recede from the teeth ; and, in the pockets thus formed, opportunity is afforded for the lodgment of particles of food and the decomposing secretions of the mouth. The ulceration, as stated on a previous page, begins along the free edge of the gums, and spreads thence superficially, rather than in depth. The entire breadth of the gums may be involved, and in severe cases the floor of the mouth may be invaded. The teeth loosen, and sometimes drop out. The gums bleed at slight touch. Salivation is free. The breath is horribly offensive. Pain is severe, especially when taking nourishment. The submaxillary lymphatics are enlarged.

Ulcerative stomatitis usually subsides entirely, under proper treatment, inside of ten days.

5. Gangrenous Stomatitis; Noma.

A constitution undermined by privation and bad hygienic surroundings seems to be a necessary predisposing factor in the production of gangrenous stomatitis. The disease usually follows some acute or chronic affection —measles and typhoid fever, in most instances. The majority of cases occur in children between the ages of two and five years. The damp seasons of the year and low-lying countries are favoring causes. Inasmuch as several cases have occurred simultaneously or near together in hospitals and families, it has been suggested that the disease is contagious.

The first objective sign is found in a small bleb on the inner surface of one cheek. This soon ruptures, and the affected part becomes indurated. Ulceration and rapid destruction of tissue follows. This gangrenous process proceeds with great rapidity, so that at the end of thirty-six hours the soft parts may be perforated. The ulceration not infrequently extends to the tongue, lips, eyes and ears. Hæmorrhage rarely takes place, because the blood-vessels are plugged previous to the separation of the sloughs. Notwithstanding the seriousness of the local manifestation, subjective symptoms are slight. Pain is noteworthy because of its absence. The breath is horribly offensive. Profound prostration is an early symptom. The prognosis is highly unfavorable, death taking place by exhaustion, the patient suffering, in the meantime, with delirium and diarrhœa.

The Gums.

Alterations in the color of the gums, as pallor, blueness, increased redness, etc., are possessed of the same diagnostic significance as similar changes in other portions of the mouth.

Increased redness, with swelling and pain, is indicative of inflammation, *i.e.*, gingivitis.

Swelling of the gums, with bluish-red discoloration, spongy appearance, and tendency to bleed on slight manipulation, is significant of *scurvy* or *scorbutus*. Sometimes ulcers are observed along the line of the teeth. The lesions are especially prominent on portions of the gums about decayed teeth. In some cases the gingival changes are limited to a circumscribed area. There is, as a rule, not much fœtor of the breath. It is important to bear in mind that this affection may occur in infants who are fed almost entirely on patented foods. In them, however, the gingival condition is not likely to appear as one of the early phenomena, but is preceded by fusiform swelling of the thighs, pains in the extremities, general malnutrition, and even by hæmaturia, although the last-named symptom is unusual excepting in severe cases.

Swelling of the gums, with spongy appearance of the same and increased flow of saliva, and possibly ulceration and stomatitis, is observed as a symptom of *mercurial poisoning*.

A red line along the dental edge of the gums was for a long time regarded as a symptom of incipient pulmonary tuberculosis. It is now recognized that it has no such important diagnostic value, and that it is found in other cachectic disorders, as diabetes and cancer.

A blue line along the free edge of the gums where they join the teeth is very characteristic of lead-poisoning. When indistinct, it may be made more prominent by the administration of iodide of potassium. It does not appear in cases of saturnism when the patients observe reasonable care in keeping the mouth clean. It is liable to persist for two or three months after the other symptoms of lead-poisoning have disappeared. It is the result of the combination of the sulphur in the secretions of the mouth with the lead deposited in the substance of the gums.

Thin yellowish or thick brown crusts may be deposited on the gums and teeth in typhoid conditions generally, and are known as *sordes*. They are composed of various micro-organisms, particles of food, and epithelial *débris*. Their component parts, it is well recognized, occur in the normal mouth; but in cases of profound asthenia, as in the typhoid state, the motions of the mouth necessary for their removal do not take place; hence their accumulation.

Receding of the gums from the teeth takes place in scurvy, stomatitis, gingivitis, and pyorrhœa alveolaris. The condition last named is an exceedingly common one, and is very destructive, so far as the teeth are concerned. Examination with a delicate probe discloses the fact that little pockets form between the gums and the teeth, and these exude pus on pressure. Pyorrhœa alveolaris is regarded by dentists generally at the present time as strongly suggestive of the uric acid diathesis. It certainly seems to have no relation to decay of the teeth, for it occurs in the gums adjoining teeth which are perfectly sound. Ultimately the teeth loosen and fall out. At times the affection is attended by considerable pain, owing to the exposure of the peridental membrane, this condition ofttimes necessitating early extraction. Pyorrhœa alveolaris may well be called the opprobrium of the dental profession, as treatment directed to its cure is generally unavailing.

Localized swelling of the gums may be due to abscesses, so-called "gum-boils," and tumors (epulides).

Gum-boils, alveolar abscesses or dental abscesses find their origin in the alveolar cavities of decayed teeth, infection taking place through the corresponding pulp-cavity, or between the alveolus and fang of a sound but dead tooth. Sometimes they form about an erupting wisdom-tooth. The pus makes its way in the direction of least resistance through the outer wall of the affected alveolus, and appears on the gums or burrows into the tissues of the face, and may thus be discharged at a distance from the original seat of disease. The condition is attended at first by a sensation as if the teeth were too large for their sockets, owing to periosteal swelling, followed soon by severe pain. The latter is greatly relieved as soon as the pus appears externally.

The various tumors of the gums are generally known as epulides. Microscopically they are found to be fibroid or myeloid growths. They spring from the alveoli, generally of the lower jaw, and are manifested as smooth, round, lobulated masses. They may ulcerate, but they never infiltrate the surrounding tissues. They commonly present between two teeth. Their growth is not very rapid.

Sometimes fibrous growths come away attached to the fangs of an extracted tooth, as if they had originated in the fibrous covering of the fang.

Nævoid conditions of the gums are occasionally observed as a local condition.

The Teeth.

An understanding of the teeth as clinical factors demands a knowledge of the time of life at which they appear. The first set of teeth, known generally as the "milk-teeth," are twenty in number, and appear as follows :

4 central incisors at about the seventh month.
4 lateral incisors at about the ninth month.
4 canines at about the eighteenth month.
4 first molars at about the twelfth month.
4 second molars at about the eighteenth month.

The second or permanent set of teeth are thirty-two in number, and appear as follows:

4 anterior molars at about the sixth year.
4 central incisors at about the seventh year.
4 lateral incisors at about the eighth year.
4 anterior bicuspids at about the ninth year.
4 posterior bicuspids at about the tenth year.
4 canines at from the eleventh to the twelfth year.
4 second molars at from the twelfth to the fourteenth year.
4 third molars, or wisdom-teeth, at from the seventeenth to the twenty-fifth year.

The eruption of the first set of teeth may be delayed or accelerated. Delayed dentition may result from ill-health from any cause, but is especially apt to arise from rachitis. Early dentition is said by Bury to be an occasional phenomenon of tuberculous tendencies.

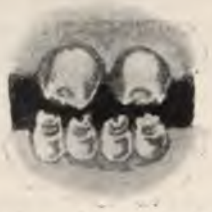
This seems to be the most appropriate place to discuss dentition as a cause of various illnesses in infants. Undoubtedly the eruption of the milk-teeth is productive, at times, of considerable disturbance. At the same time, it should be remembered that it has been vastly overrated as a cause of disease. The various ailments which have been alleged to result from dentition would require a large volume for their description, while those that have been ignored in this respect could be described on one's thumb-nail. Naturally, some local discomfort is not unusual as an attendant upon the eruption of the teeth, and this may readily excite a certain degree of peevishness on the part of the child. In highly-neurotic children this irritation may excite reflex convulsions. The tender mouth may lead to either insufficient nourishment or the retention of the food in the mouth for too short a time; hence disorders of the gastro-intestinal tract may result. These, I believe, constitute the sole effects of difficult dentition, *per se*. In constitutional conditions, the eruption of the teeth may not proceed normally; but in such cases the associated symptoms are far more likely to be an expression of the general disease than of the gingival and dental irritation. The treatment of such cases must be conducted on a constitutional basis, even though local measures may relieve the mouth-symptoms and hasten the eruption of the teeth.

Deformities of the Teeth.—These include crowding of the teeth, irregular growth and misplacement, furrowing of the teeth, pitted and rocky enamel, and syphilitic teeth.

Crowding, irregular growth and *displacement* have but little bearing on clinical medicine, as their chief relief is the duty of the dentist. Their presence, however, is occasionally an important factor in the production of disorder elsewhere, mainly in the digestive processes. This statement applies also to affections of the teeth in general. Good digestion is not likely to exist if the teeth are not sound; and if the digestion is poor, the general nutrition of the body is never up to the normal standard, and thus numerous ills may come.

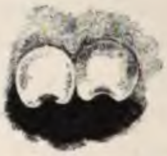
Furrowing of the teeth is generally observed as horizontal lines. Proper examination discloses that these lines correspond exactly on teeth which have been erupted at about the same time. It is fair to presume, therefore, that the deformity is the result of causes producing general or local ill-health during the predental period. Jonathan Hutchinson taught at one time that furrowing resulted from the administration of mercury to infants. It has since been demonstrated that it is of frequent occurrence in infants who have never taken mercury in any of its forms, and that it is not infrequent in those who have had any one of the varieties of stomatitis, or who have been seriously ill from any cause.

FIG. 38.



The upper incisors show incomplete notches; they diverge, leaving a wide interspace. The lower incisors present a series of small excrescences.—After Hutchinson.

FIG. 39.



Upper central incisors showing the characteristic central notch. They are narrow at their edges and slope towards each other.—After Hutchinson.

Irregularity of form, stunting or *pitting of the teeth* may occur in rachitis or syphilitic children, and in idiots. Such teeth soon become carious. This condition is not to be regarded as of diagnostic value. It is simply a local expression of a constitutional state. Periostitis and gingivitis may be set up as a secondary condition.

A peculiar notching of the incisors of the permanent set of teeth occurs as one of the results of hereditary syphilis. The teeth affected are characteristically the upper central incisors, but the lateral incisors and the canines may also be involved. This deformity of the teeth was first described by Jonathan Hutchinson, hence it is commonly known as the "*Hutchinson teeth.*" The affected structures are peg-shaped, their bases being broader than their cutting-edges, and along the latter will be found a single semilunar-shaped notch. If such teeth are associated with symmetrical deafness and interstitial keratitis, the diagnosis of hereditary syphilis may be regarded as certain. These three symptoms are sometimes spoken of as "Hutchinson's triad of symptoms."

Caries of the teeth is favored by certain constitutional conditions. It results from local causes, mainly. Some of these are defective structure of enamel and dentine and irregular growth favoring the lodgment and decomposition of food, want of cleanliness, and the taking of such medicines as iron, mercury, etc.

Loosening of the teeth occurs as a result of prolonged administration of mercury, iodide of potassium, scurvy, gangrenous stomatitis, and diabetes.

Pain is a common symptom of dental disorders, of which caries is the most frequently observed. It must be borne in mind, however, that although the teeth are apparently sound, these structures may still be the cause of pain. Dead teeth with decomposed pulp, periodontitis (pericementitis, periostitis), pyorrhœa alveolaris, and other affections, must be kept in mind.

Grinding of the teeth is commonly observed in highly-neurotic children during sleep. In the majority of cases it is due to gastro-intestinal disorders, though it may occasionally be associated with disease of the brain. It must not be regarded as symptomatic of "worms," although it does occur sometimes when intestinal parasites are present. It is then, as at other times, an expression of the gastro-intestinal disorder.

The Tongue.

Current views respecting the clinical value of the appearances of the tongue are sadly in need of revision. It is not that accurate knowledge concerning the subject is wanting, but that the study of this organ in the investigation of disease is surrounded with altogether too much superstitious reverence. Great importance has been attached in the past to this or that appearance of the tongue as diagnostic of this or that particular disease, when in reality the tongue scarcely ever indicates the existence of a definite pathological condition, but is merely an index of the constitutional state. It is to be regretted that the laity is mistakenly impressed with the value of the tongue in diagnosis, thus causing needless worry in many instances in which a coated tongue happens to be the only evidence of ill-health. More than that, some are offended if the inspection of the tongue is not performed with all the reverence of a religious ceremony. The writer believes that all clinical phenomena are explainable on scientific physiological and pathological principles, and that the conditions of the tongue in disease are no exception to this opinion. Acceptance of the following teachings does not make the tongue an unimportant factor in diagnosis and treatment, but it puts the indications afforded by inspection of the organ on a reliable foundation.

When-examining the tongue, the physician should note:

1. Its size.
2. Its shape.
3. Its color.
4. Its degree of moisture.
5. Its movements.
6. Its coatings.
7. Its surface as to the presence or absence of fissures, ulcerations, eruptions and elevations.

1. **The Size of the Tongue.**—The tongue may, by reason of disease, be either enlarged or diminished in size. It is enlarged in

a. Glossitis.
b. Hemiglossitis.
c. Macroglossia.
d. Myxœdema.
e. Chronic heart disease.
f. Anæmia.
g. Liver disease.
h. Mercurial poisoning.
i. Cancer.
j. Gummata. } Enlargements circumscribed.
k. Cysts.

(*a*) **Glossitis.**—In this disease the enlargement is due to inflammatory swelling, and may be so great that the tongue becomes two or three times the normal size. In extreme cases it may so fill up the cavity of the mouth as to produce obstructive dyspnœa. Resolution usually takes place in three or four days. The disease may, however, terminate in gangrene or suppuration.

(*b*) **Hemiglossitis.**—This is characterized by limitation of the inflammatory swelling to one lateral half of the tongue. Its symptoms are by no means as severe as those of glossitis. It may, however, terminate in suppuration. The varieties of glossitis to which reference has been made above invade the body or substance of the tongue. The mucous membrane alone is frequently involved in cases of stomatitis, and may be designated "superficial glossitis," the swelling in such cases never being very prominent. There is, however, a form of chronic superficial glossitis which runs a drawn-out course, and, while sometimes due to excessive use of tobacco, is oftener traceable to gastric disorder in rheumatic subjects, with local irritation as an exciting cause.

Inflammation of the tongue is due, as a rule, to traumatism and infection. Other causes have been alleged, as exposure to cold, alcoholic excesses, mercurial poisoning, and the acute specific infections—notably typhoid fever.

(*c*) **Macroglossia,** or enlarged tongue, is usually a congenital affection, and seems to be comparatively common in idiots and others mentally deficient.

(*d*) **Myxœdema,** in which disease the tongue is swollen by reason of the same mucoid infiltration that invades other portions of the body in this disease.

(*e*) In **chronic heart disease** the lingual enlargement results from the venous engorgement of the organ. In this connection it is well to observe that any general condition of the system attended by sluggishness of the circulation may present enlargement of the tongue,—not necessarily, however, to a marked degree.

(*f*) In **anæmia** we find the tongue both large and flabby, owing to the participation of its tissues in the general relaxation. The anæmic tongue usually shows the imprint of the teeth, although this symptom is not apt to be as prominent a feature as in the enlarged tongue of liver disease and mercurial poisoning.

(*g*) **Liver disease** is often associated with enlargement of the tongue, though not to a remarkable extent. Characteristically, the tongue takes the imprint of the teeth. The reader is cautioned against regarding this symptom as pathognomonic.

(*h*) **Mercurial poisoning** has enlargement of the tongue, which takes the imprint of the teeth ; but there are also associated salivation and sponginess of the gums.

(*i*) **Cancer.**—Cancer of the tongue is practically always an epithelioma. It may start as ulceration, a wart, or a tumor. Even when taking the last-mentioned form in the beginning, the physical conditions of the mouth favor rapid ulceration, the characteristics of which will be considered under ulcerations of the tongue. The age of the patient is an important diagnostic point. The usual though not the invariable site is the edge of the middle-third of the tongue. It starts as an induration, which the patient describes as a lump, but which may have already undergone degeneration when first seen by the physician. Sometimes papillary masses are seen to project from its surface. In such cases ulceration is not prominent, nor is the induration so great. The pathological process may extend to neighboring structures, limiting the movements of the organ. The submaxillary lymphatics are invaded. Epithelioma of the tongue is especially liable to be confounded with

(*j*) **Gummata,** which will present, if the patient is truthful, a syphilitic history. The overlying mucous surface is smooth, and lacks the well-defined outline and induration of the malignant growth. Like cancer, gummata are exceedingly prone to ulceration.

(*k*) **Cystic growths of the tongue** include dermoid, mucous and blood cysts. (i) Lingual dermoids, while of congenital origin, are not always by any means sufficiently large at birth to force their recognition. Indeed, they may remain small for many years. In their characteristic form they are smooth, elastic, round or oval. They are situated beneath the tongue, projecting rather towards its surface than into its substance. (ii) Mucous cysts are due to dilatation of the mucous glands, and are liable to appear in those places where mucous glands are the most numerous, *i.e.*, at the base of the tongue. They consist of tense elastic fluctuating swellings, well-circumscribed. The overlying mucous membrane is normal. They cannot be mistaken for anything but abscess, from which they are to be differentiated by the absence of pain and the examination of their contents.

2. **The Tongue is small** in

a. Peritonitis.
b. Chronic gastritis.
c. Atrophy of its muscular substance.

The tongue is generally spoken of as small when it is reduced in size without any wasting of its tissues. This use of the term must be regarded as convenient, not scientific. We may also speak of atrophy, in which case the muscular substance of the tongue is especially involved. The principles underlying atrophy of the lingual muscles are the same as those producing atrophy of muscular tissue elsewhere. (*Vide* Muscular Atrophy.) The lesions in which atrophy occurs are :

(*i*) Injuries or diseases of the hypoglossal nerves or their nuclei.
(*ii*) Glosso-labio-laryngeal or bulbar paralysis.
(*iii*) Atrophy of disuse in old hemiplegias.

In (*i*) and (*ii*) the atrophy is numerical.

3. **Color of the Tongue.**—Alterations in the color of the tongue afford indications as follows :

Pale in anæmia.

Cyanotic or **bluish-red** in heart diseases with failing compensation. Examination of the under-surface of tongue will in such cases show dilatation of the sublingual veins.

Purplish or **blackish spots** on the tongue are not uncommon in Addison's disease. To be diagnostic, however, they must be associated with a bronzing of the skin. They have sharply-defined borders, and are on a level with the surrounding mucous membrane.

Reddish, later becoming bluish to black submucous blotches, indicate hæmorrhage into the submucous tissues. They may be the result of purpura hæmorrhagica, and in that case are not infrequently associated with bluish-black discolorations, looking like bruises, on various portions of the body ; or they may have been produced by traumatism. These extravasations, when fresh and of a bright color, may be differentiated from congestion and inflammation by pressure, which does not remove the discoloration of extravasations.

Staining of the tongue may occur from various substances: *e.g.*, (i) *black stains* are caused by biniodide of mercury, the various bismuth preparations, and iron; (ii) *yellow stains* by tobacco; (iii) *yellowish-brown* by licorice; (iv) *orange-yellow staining* is common among tea-tasters.

Yellow patches, oblong in shape, slightly raised above the surrounding mucous membrane and with well-defined borders, ranging in size from an eighth to a third of an inch in diameter, constitute *xanthelasma*. Similar lesions may be found also on the eyelids, palms of the hands, and other portions of the body.

Abnormal redness of the tongue may be observed in the early stages of most febrile and inflammatory affections.

Dark-red, merging almost into a black, may be observed in typhus and typhoid fevers, and in typhoid conditions generally.

Crimson-red and **red coloration** of the tongue may be observed in high degrees of gastro-intestinal irritation and diabetes. The raw, red, denuded and dry tongue is strongly indicative of great depression of the vital forces.

Black tongue is symptomatic of a rare parasitic affection, known as *nigrities*. The discoloration is usually situated in the middle of the dorsum of the tongue. The papillæ of the affected part are elongated. Beginning as a small spot, it spreads until the entire organ is involved, the discoloration being most pronounced at the initial point. When it disappears, it does so by paling at the periphery, and is followed by desquamation.

4. **Degree of Moisture of the Tongue.**—Moisture and dryness should receive especial attention during the examination of that organ, for an unnaturally dry tongue, if not due to accidental or physiological causes, may be regarded as having an unfavorable significance, while a dry tongue becoming moist is correspondingly favorable. Occurring in the course of acute diseases, dry tongue is a certain indication for liquid diet; occurring in chronic disorders, it augurs an unfavorable issue. Physiological dryness of the mouth and tongue is a common thing in mouth-breathers. Pathologically, it is observed in the course of all fevers, its presence being favored more by long duration than by the severity of the fever. It is common in all diseases attended by dehydration of the body, *e.g.*, diabetes and diarrhœa. A notable exception to this last statement is found in cholera Asiatica, in which disease, notwithstanding the profuse vomiting and purging, the tongue remains moist. This anomaly has been explained by the constant moistening of the mouth by the watery vomit. But this explanation is not satisfactory, because moistening of a dry mouth is but short-lasting in its effects. The moist mouth of cholera is probably due to the low bodily temperature, for when reaction sets in, and the discharges have ceased and fever appears, the tongue becomes dry. Chronic

diseases with dryness of the tongue are characterized, for the most part, by prostration and exhaustion.

5. **Movements of the Tongue.**—When studying the movements of the tongue, note whether it is protruded slowly or quickly, when requested; whether its movements are steady or tremulous; whether the tongue can be moved at all; or whether it is the subject of abnormal movements (spasms or restless movements). In all cases, before attaching any importance to the results of the examination, allow for the temperament of the individual.

The tongue is protruded *quickly* in hysteria and hypochondriasis—almost as if the act afforded the patient great pleasure.

It is protruded *slowly* in typhoid condition and in patients whose mental condition is blunted, as in beginning stupor.

Tremulousness of the tongue is observed in general asthenia and general paralysis of the insane. Asthenic tremor is common in severe cases of typhoid fever, and is said to indicate deep intestinal ulceration. In general paralysis of the insane the lingual tremor is associated with tremor of the lips and the mental and physical symptoms of that disease.

Paralysis of the tongue may be either bilateral or unilateral, and with or without atrophy of that organ. Unilateral paralysis of the tongue without atrophy (excepting the slight degree which may result from prolonged disuse) is always indicative of a cerebral lesion. In bilateral paralysis of the tongue, that organ lies motionless on the floor of the mouth. In the case of unilateral paralysis, the tongue while at rest deviates from the paralyzed side; but, when protruded, towards the paralyzed side. Paralysis of the tongue is observed in

a. Brain affections of all kinds, especially, however, in apoplexy.
b. Disease or injury of one or both hypoglossal nerves.
c. Glosso-labio-laryngeal or bulbar palsy.
d. Disseminated sclerosis.
e. Post-diphtheritic paralysis.

In tongues undergoing atrophy minute fibrillary twitchings are readily observable, and indicate that the atrophic process is still active.

Spasm of the tongue may occur in

a. Chorea.
b. Hysteria.
c. Epilepsy.
d. Irritation of the hypoglossal nerves.
e. Thomsen's disease.
f. Disseminated sclerosis.

The spasm of the tongue in chorea constitutes one of the expressions of muscular unrest so prevalent throughout the body in that disease. Commonly it only interferes with speech, but it may be so severe as to render it impossible for the patient to speak intelligibly. Spasm of the tongue in hysteria is rare, excepting as part of a hysterical seizure. No matter how severe the convulsion, the tongue is never bitten. The lingual spasms of epilepsy are likewise part of the general paroxysms. Severe biting of the tongue is characteristic and quite common in epilepsy.

As the result of irritation of the fifth nerve, spasm of the tongue may arise from carious teeth, especially when the latter are so situated as to produce lingual ulceration.

Idiopathic spasm of the tongue, apparently of the same character as *tic convulsif* of the face, has been reported. Sometimes it is associated with a similar facial spasm.

In a case reported by Fleury, spasm of the tongue was excited by attempts at speaking. He denominated the affection "aphthongia," and regarded it as an affection allied to writer's cramp.

6. **Coating of the Tongue.**—It is especially in relation to this particular subject that there has been much erroneous teaching. The investigations of Dickinson, conducted as they were with every attention to scientific detail, are therefore invaluable. This authority made careful microscopical studies of the coatings on many tongues, and studied the findings in relation to the various factors entering into the clinical pictures exhibited by the patients. He presented a classification of tongues easy of comprehension, and readily applied in clinical work.

The coating of the tongue was found by microscopical examinations to consist of accumulations of epithelial and food *débris*, fat-globules, amorphous matter, and micro-organisms. Regular washing of the mouth and the movements of the tongue prevent the coating from becoming prominent in health. If, from any cause, the movements of the tongue are restricted in whole or in part, that organ will be coated accordingly. Mechanical causes, however, are not the only ones influencing coating. The rate of epithelial growth is a most important factor. With some individuals this is manifestly much greater than in others; hence such persons are more likely to present a coated tongue. Then, too, pyrexia has been found to favor an epithelial overgrowth. An additional factor is found in the readiness with which some coatings disappear under the mechanical effect of movement of the tongue. If that organ is dry, the epithelium is hardened and becomes more resistant to friction, and hence the tendency to accumulation is a marked one and the coating becomes correspondingly thick. With these preliminary remarks the classification of tongues as proposed by Dickinson is herewith presented, together with the microscopical findings in each, and the relation to constitutional states.

(*a*) The **Stippled tongue,** characterized by a coating which appears as minute white spots scattered over the surface of the tongue. Microscopically, it is found to consist of white epithelium in small amount on papillæ, not continuous or superabundant. Such a tongue is mainly the evidence of disuse. It seldom occurs where there is much prostration or grave constitutional disturbance or pyrexia. The stippled tongue may become dry, in which case its relation to constitutional disturbance is evident.

(*b*) **Stippled plus coated tongue,** in which the minute white dots above described are present; but, in addition, there is a general whiteness, as if there had been a deposit of "frost." It has been found, microscopically, to be dependent upon white epithelium in excess on the papillæ, with partial filling of the intervals. This variety of tongue is present in a large proportion of patients with chronic disease, but is more apt than the preceding to be found in acute and constitutional affections. Pyrexia is not unusual, and the constitutional state of very many of the patients is such as to demand a liquid diet. When dryness is an additional clinical feature, these indications become still stronger in their significance.

(*c*) **Coated tongue,** in which the coating is even and continuous, and thicker than in the preceding. It is due to excess of white epithelium on the papillæ; intervals more or less filled up with epithelium and accidental matter. Clinically, the coated tongue brings with it a further increase of acute and febrile disease. Pyrexia and prostration are both on the ascent, showing that the generally coated tongue is associated with a larger proportion of constitutional affections than where the coat is partial. The tendency to dryness is also greater, this adding force to the indications.

The coating may disappear in one of two ways: "It may gradually thin off with a shelving edge, exposing a moist and natural surface, or it may break away in flakes after displaying a surface which is red and dry. The former quotation, implying, as it usually does, the restoration of the healthy surface, is obviously of better omen."

(*d*) **Strawberry tongue,** which is described by Dickinson as but a variety of the coated tongue. In this the fungiform papillæ show through the coating, especially at the tip and edges. The papillæ are more or less injected, and hence are rendered prominent. This tongue is commonly observed in scarlatina, though also seen occasionally in pneumonia, typhoid fever and appendicitis. Pyrexia is commonly present in cases of strawberry tongue.

(*e*) **Plastered Tongue.**—This presents a uniform thick, white coating, suggesting, almost, that it has been laid on with a trowel. The microscope shows more elongation of the papillæ than with the coated tongue, and more filling of intervals with superficial accumulation. The plastered tongue is the tongue of acute disease. When the mucous surface of the tongue is exposed in any part, it is found to be redder than normal. The more recent the illness, the shorter the coat. The thickness of the coating is a good index of the severity of the disease, for illness is an important factor in the rapidity of the accumulation. With duration or chronicity, the tendency is to become brown or dirty-yellowish. Prostration is usually associated with the pyrexia when this tongue is present.

(*f*) The **furred and shaggy tongue**, characterized by extravagantly-long papillæ, mostly of horny epithelium. It is largely the result of disuse and want of moisture, the latter preventing a proper wearing away of the epithelial surface. It is often observed in old age, and in cases of constipation. It is suggestive of failure of nutrition and waning of vital powers.

(*g*) **Encrusted Dry and Brown Tongue.**—The tongue is covered with a dry, brown crust, which surmounts and conceals the papillæ, and is often broken and fissured. Microscopically, there is found a continuous crust on and between the papillæ, consisting largely of parasitic matters. The brownish discoloration is due to dryness; for, as Dickinson remarks, many animal matters dry brown, although, in this case, the staining of the coating by food and medicine may aid somewhat. This tongue may be regarded as strong evidence of prostration and depression. It is found in cases with lowered vitality, as phthisis, cancer, typhoid fever, and failure of circulation. It is very common in cases of coma.

(*h*) **Denuded red tongue**, characterized by absence of the normal epithelial covering, and dependent, as shown by the microscope, upon general absence of all excepting the Malpighian layer—sometimes of that also. It occurs generally in conditions of exhaustion. It is a frequent concomitant of diseases having exhausting discharges, especially diarrhœa and suppuration. It is more apt to occur in chronic diseases than the dry, encrusted tongue. It is observed frequently in dysentery, especially when complicated with abscess of the liver. The salivary secretion is always deficient. It is produced by impairment of the epithelial growth and want of saliva, and irritation of the mucous membrane by the contents of the mouth. With improvement in the condition of the patient the redness fades, the papillæ become softer, and moisture returns.

(*i*) The **cyanosed tongue** presents to the naked eye a bluish or purple color, and a "smooth, wet, slippery surface, like that of an eel, upon which the papillæ are almost indistinguishable, as if fused together or in some way deprived of their separating intervals. Upon this substratum may be overlaid more or less of one of the grades of coat which have been described, to the partial obscuring of the deeper characters. Microscopically, the most noticeable peculiarities are superabundance of the recent epithelium, vascular injection, and hypernucleation." It occurs in heart diseases and in albuminuria.

7. **Fissures and Furrows.**—Fissures or furrows of the tongue may be single or multiple. In the former case the lesion is generally observed occupying the median line. Here there is a natural furrow, which tends to become irritated on slight provocation, its edges being

deprived of both papillæ and fur. At the same time it must be borne in mind that a fissure may occupy any portion of the organ. Multiple fissures may be transverse, longitudinal or diagonal, interlacing each other in every conceivable way. Furrowing of the tongue may be observed in many healthy persons, especially in those accustomed to the use of alcohol and tobacco. It is not uncommon in the aged, in febrile affections, in digestive disorders, and as a result of past lingual inflammation.

Very deep furrows constitute one of the manifestations of a condition described by Wunderlich as *dissecting glossitis*, and is an aggravated form of superficial glossitis. It is sometimes the result of syphilis.

Fissures may be caused by irritation from the broken or rough edge of a tooth. In such cases the floor of the lesion will be found ulcerated, and its cause made manifest by its contiguity to the abnormal tooth.

Fissures also occur during the course of secondary syphilis, their localization being favored by irritation. Hence they appear where the tongue comes in contact with a rough tooth-edge or hot tobacco-smoke. They are not apt to be painful unless the surrounding structure is inflamed secondarily.

Carcinomatous fissures are always secondary to a distinct tumor, and there is involvement of the submaxillary lymphatics.

Tubercular fissures occur only in association with evidences of tuberculosis in other portions of the body. Beginning as an ulcer, or as a small and shallow cleft, the lesions increase in number and depth, or become stellate or irregularly branched. The lymphatic glands are always involved.

8. **Ulcerations.**—These may be simple, aphthous, traumatic, dyspeptic, syphilitic, carcinomatous, and tubercular.

The so-called *simple ulcers* are the results of chronic glossitis. They are usually situated on the centre of the tongue; have a smooth, red, glazed surface; are irregular in outline, and have indolent, indurated edges. They are painful.

Aphthous ulcers of the tongue possess the same features as similar ulcers of the mouth, and are especially common in children, though by no means limited to them.

Dyspeptic ulcers are small in size, and are situated on or near the tip of the tongue. The surrounding tissue is quite red. They are very painful, and exhibit a remarkable tendency to recurrence.

Tubercular ulceration is practically always associated with evidences of tuberculosis elsewhere. The ulcers are irregular in shape, often stellate; are covered by grayish, purulent or yellow discharge; are not surrounded by inflammatory changes, but are exceedingly painful. There is always involvement of the submaxillary lymphatics, herein presenting a very important point for differentiation from secondary syphilitic ulcerations, which never have such glandular complications. The tubercular ulcerations have sharp-cut or bevelled edges. Musser remarks that in their neighborhood "a number of tiny, yellowish-gray points may be observed. The ulcer is painful, and attended by salivation."

Syphilitic ulcers usually succeed some other lesion, as papules, gummata, and patches. They are generally painless, unless there is a secondary inflammation, and are usually multiple. The tertiary varieties are noteworthy for the depth to which they may extend. They are apt to be followed by sclerosis of the tongue. (Fournier.)

The **carcinomatous ulcer** is preceded by a distinct tumor.

9. **The Sense of Taste.**—The tongue being the principal organ of taste, the study of alterations in this function may be best taken up in this connection. Diminution or absence of the sense of taste is known as *ageusia;* hyperæsthesia of the sense of taste as *hypergeusia;* and alterations in the sense of taste as *parageusia.*

When examining the condition of the gustatory sense, we test only for sweet, sour, salty, and bitter sensations. This may be done by applying to the tongue, in turn, sugar, vinegar, salt and quinine. The tongue is to be protruded. Care should be observed that it is not permitted to touch the lips or to be drawn back into the mouth in the course of the experiment. The test-substance is applied, in turn, to the back of the tongue, first on one side, and then on the other; next, the anterior portion of the tongue is to be examined in the same manner. As there are some gustatory fibres in the soft palate and mucous membrane of the lips, these parts should also be examined. The test for bitter (quinine) should be applied last, as, otherwise, it will interfere with the taste of other substances.

Ageusia may be due to local conditions about the mouth, or to disturbances of the gustatory sensory apparatus. Of the local causes may be mentioned the dry mouth of fevers, catarrhal inflammation of the buccal mucous membrane, and all conditions in which the tongue is heavily coated. As evidence of a nerve lesion, it occurs in facial neuritis with involvement of the chorda tympani nerve, hysteria, and in diseases of the trigeminal and glosso-pharyngeal nerves. It has been reported as of bulbar origin, then being due to nuclear degeneration, especially in association with tabes dorsalis. In hysteria it is apt to be associated with hemianæsthesia.

It is well known that delicate flavors are detected by the olfactory sense. Hence it is that in catarrhal rhinitis and other diseases of the nasal passages defective sense of taste is commonly observed.

Parageusia.—Perverted sense of taste may be one of the symptoms of hysteria or insanity. It may also occur in gastric disorders.

Morbid gustatory sensations may be experienced by reason of the presence of abnormal substances in solution in the saliva. Thus, persons taking iodide of potassium often complain of a brassy taste. The foul taste in the mouth experienced by sufferers from dyspepsia, jaundice, lithæmia, carious teeth, etc., is probably dependent upon the accumulation of epithelial *débris* in the mouth, and the elimination of toxines by the saliva.

The Lingual Tonsil and Its Lesions.

Widely-divergent views respecting disease of the lingual tonsil prevail. One practically ignores the subject; the other, which is so strongly upheld by Lennox Browne, holds that lesions in this locality are very common, and are productive of great discomfort. As in most such instances, the truth lies between the extremes. The diseases which may involve the lingual tonsil include catarrhal inflammation, abscess, hypertrophy, and varicose veins.

Catarrhal Inflammation of the Lingual Tonsil.—The occurrence of this condition independently of inflammation of the faucial tonsils is rare. Subjectively, the symptoms include the sensation as of a foreign body which forces the patient to make frequent ineffectual attempts at swallowing. The patient refers the sensation externally to the level of the hyoid bone. Inspection of the throat shows only swelling and redness, and swelling of the lower portion of the anterior pillar of the fauces where it joins the root of the tongue. The diagnosis cannot be established until the lingual tonsil has been inspected by the mirror, when it will appear swollen and inflamed, with accumulation of secretions in its crypts. The prognosis is favorable, but the disease is apt to last for two or three weeks.

Abscess of the Lingual Tonsil.—This, likewise, is a very rare disease, and, moreover, a serious one. It is stated that the cases of death during the course of quinsy are in reality dependent upon abscess of the lingual tonsil as a complication. The symptoms of abscess of the lingual tonsil include severe pain not only during the acts of swallowing, but also during the intervals. Fever is high. The swelling of the affected parts is usually sufficient to present a mechanical impediment to the passage of food and to easy respiration. In some cases the inflammation extends to adjacent structures, as the larynx and floor of the mouth. Considerable danger attends this disease, owing to possible suffocation when the abscess ruptures.

Varicose Veins of the Lingual Tonsil.—As to the frequency with which varicose veins may be discovered in this location there can be no doubt, but whether such a lesion is capable of producing any serious disturbance is a debatable question. Lennox Browne regards them as of serious importance, claiming that they may give rise to pains, morbid sensations at the base of the tongue or in the throat (globus hystericus), torticollis, abnormal vocalization, cough, hemming, dyspnœa, dysphagia, faucial tenesmus, and hæmorrhage. The diagnosis can only be made by laryngoscopic examination.

The Saliva.

The saliva is a glairy, opalescent fluid, having a specific gravity ranging from 1002 to 1008. It contains albumin,—ptyalin, mainly. Its chemical reaction is alkaline. The importance of this fluid in the digestive process is greatly neglected. Its ptyalin has the property of converting hydrated starch into glucose; in other words, it exerts a diastasic action. Hence, certain foods are partially digested while still in the mouth, if mastication be properly prolonged. As the result of disease, the saliva may be either increased or diminished in quantity, or its chemical reaction altered.

The salivary flow is increased in

(*a*) **All mouth and throat inflammations**; especially so in the different varieties of stomatitis. In ulcerative stomatitis it is furthermore altered in color by reason of the contained leucocytes, blood-corpuscles, and degenerated epithelium.

(*b*) **Pregnancy.**—This particular form of salivation is of considerable practical interest, because of the great annoyance it causes when at all marked. Therapeutically, it is one of the opprobriums of the profession. The increased flow in some cases is truly remarkable, being four to six times the normal quantity. It is especially liable to occur in neurotic subjects, and may be associated with nephritis.

(*c*) **Certain nervous affections**; notably in acute mania and hydrophobia.

(*d*) **Certain drugs,** as mercury and pilocarpine, produce salivation. The salivation of the former is attended by the characteristic mercurial sore mouth, and that of the latter by increased secretion of all the mucous membranes and profuse perspiration.

(*e*) **Some Cases of Chronic Gastritis.**

The salivary flow is diminished in

a. Fever.
b. Diabetes.
c. Diarrhœa.
d. After certain drugs, as atropia.
e. Diseases of the salivary glands, notably of the parotid gland.

For a further consideration of diminished salivary flow, the reader is referred to the paragraphs on dryness of the mouth (page 153).

The clinical value of the reaction of the saliva is at present unknown. It is admitted that the saliva is acid in reaction in

a. Fevers.
b. Inflammations and ulcerations of the mouth.
c. Diabetes, gout and rheumatism.

Offensive Breath.

The causes leading to the production of offensive breath are for the most part those which favor the accumulation and decomposition of secretions, and the formation of dead tissue in the mouth and pharynx. Substances which are eliminated by the lungs impart their odor to the breath. The causes of offensive breath are as follows:

1. Catarrhal and other ulcerations of the mouth.
2. Decayed teeth.
3. Inflammation of the pharynx and tonsils, especially the acute varieties.
4. Diseases of the stomach.
5. Affections of the brain.
6. Fevers, especially if long-continued.
7. Diabetic coma, in which case the breath has a peculiar fruity odor.
8. Certain drugs, as mercury, phosphorus, opium, alcohol, onions, etc.
9. Necrosis of the jaw.
10. Respiratory diseases accompanied by ulceration, destruction of tissue, and retention of secretions.
11. Syphilitic ulceration of pharynx and larynx.
12. Unknown causes.

Fauces, Pharynx and Tonsils.

Method of Examination.—Accuracy of observation demands proper preparation for the examination; in other words, the physician should first make sure that he has the patient in such a position that he can light up the parts thoroughly. If the patient is not bedridden, have him seated near the window; if in bed, see that the room is well-lighted; or, if the patient is too far from the windows, use artificial light and the head mirror as may be convenient; but, in any event, be sure that there is a sufficiently bright light. It is also important that all obstacles to proper inspection be removed; this means that the tongue must be well depressed. If the patient cannot do this himself, then the tongue-depressor must be employed. If for any reason it is undesirable to do this, then have the patient take a deep breath or to pronounce the sound "ah," when the palate will be raised, the tongue depressed, and the entire pharynx and tonsils will be exposed to view.

In the case of infants and children, one is likely to have some trouble in exposing the parts thoroughly. Having placed the patient in a position that will afford good lighting, use one's finger as a tongue-depressor, but in so doing take care to have part of the child's lip between that finger and the teeth. This will insure against being bitten.

What to Observe.—Having the parts in view, it is next in order to bear in mind what is to be observed. Pay attention, then, to the following points:

1. The color of the mucous membrane.
2. The presence or absence of swellings of the mucous membrane of the fauces, tonsils, etc.
3. The condition of the uvula, palate, fauces, pharynx and tonsils, as to movement, shape, secretions, etc.
4. Exudations.
5. Ulcerations.
6. Elevations.
7. The condition of the vessels of the pharynx.

Preceding the examination, an inquiry into the history of the case and the sufferings of the patient constitute an important factor in that it aids quick observation and the interpretation of what one sees. The anatomical structure of the pharynx is to be borne in mind, namely: that it is a tube lined with mucous membrane, beneath which is connective tissue and muscles. Then, too, inquiry must be made respecting the pharyngeal functions,—those of deglutition and respiration.

1. **Color of the Mucous Membrane.**—*Increased redness* of the pharynx is observed in all varieties of inflammation—in some throughout their course, in others only at the beginning.

A **cyanosed hue** or lividity, due, of course, to venous congestion, occurs as the result of cardiac and pulmonary disorders, and in obstruction of the superior vena cava.

Capillary pulsation is sometimes present, and is suggestive of tricuspid regurgitation.

The various exanthemata show changes of the pharyngeal mucous membrane which may be regarded as the mucous membrane prototypes of the cutaneous condition. Thus, increased redness is observed in *scarlatina;* a patchy redness in *measles;* and papules, followed by vesiculation and pustulation, in *variola.*

Pallor of the pharynx is common in all forms of anæmia. A pallor of the pharynx, when not accompanied by other evidences of anæmia, is suggestive of a tubercular tendency.

Erythema of the pharynx is an occasional symptom in typhoid fever.

Yellowish hue is sometimes observed in this locality in jaundice, when not discoverable elsewhere.

2. **Alterations in the size of the pharynx,** which may be due to swelling, on the one hand, diminishing the lumen of the tube, and atrophy of the pharyngeal structures, giving rise to increase of the lumen or diverticula.

Swelling of the tissues—especially that of an acute character—is an attendant upon all inflammatory affections of the fauces, pharynx and tonsils. In acute catarrhal pharyngitis it is never extreme, being limited to the mucous membrane. In the deeper-seated inflammations, as the phlegmonous, the erysipelatous and gangrenous varieties, it may become of itself a source of danger.

The swelling from inflammation of the peripharyngeal connective tissue is typified in **retropharyngeal abscess** this being the most common variety, although it may take the form of latero-pharyngeal and antero-pharyngeal, due to lateral and anterior involvement respectively. The proper understanding of the evolution of these abscesses necessitates that we bear in mind the construction of the parts. The cavity back of the pharynx contains, besides the connective tissue, the superior ganglion of the sympathetic and Gillette's ganglion, the latter receiving the lymphatics of the neck, naso-pharynx, and pharynx. Inflammation can only take place through infection, the ganglia being the first to suffer in the majority of cases. The connective tissue is primarily involved when the trouble arises from a perforating infecting wound or from extension of inflammation from the pharynx itself. The constitutional causes favoring the disorder include bad hygienic surroundings, lymphatism, hereditary syphilis, and the various acute infectious diseases. It seems to occur with especial frequency in infants and young children. The swelling is nearly always unilateral, red or purplish in color, projecting out towards the buccal cavity, and may be readily seen by simple inspection. When, however, it is situated low in the pharynx, it may require the laryngoscope or the finger for its discovery. To the latter it gives a soft, doughy sensation; sometimes it is possible to detect fluctuation. The swelling interferes greatly with both deglutition and respiration.

The **latero-pharyngeal abscesses** are not so likely to occur in children as are the preceding, and are dependent upon a very different set of causes, including acute tonsillitis, acute pharyngitis, acute rhinitis, dental caries, periostitis, and ulceration of the pharynx. Inspection shows a projecting and inflamed tonsil, with surrounding inflammation and swelling of the pharyngeal wall, extending especially in a backward direction. Examination externally demonstrates a swelling of the neck corresponding to that in the throat. The natural depression beneath the angle of the jaw is replaced by a swelling which gives a boggy sensation to palpation. In some cases this swelling may be so great as to obliterate the normal contour of the neck. The overlying skin may be red and œdematous. With these local changes may be associated the regular phenomena of acute suppuration, namely, chills, fever, and prostration. Mechanically, the lesion interferes materially with deglutition and respiration. This condition may be mistaken for an adeno-phlegmon of the angle of the jaw, from which, however, it may readily be differentiated by the presence of swelling and pain in the throat itself.

Antero-pharyngeal abscess finds its location in the layer of connective tissue lying between the larynx and the pharynx. It nearly always arises from laryngeal disease. It can be recognized only by laryngoscopic examination, and appears as a swelling of the pharyngeal mucous membrane anteriorly. It interferes with both deglutition and respiration, the latter especially when the laryngeal mucous membrane becomes œdematous.

Very rarely the inflammation is not confined to one of the localities above specified, but extends in all directions, in which case the condition is known as *peripharyngeal abscess.* The swelling then is general and of rapid progress. Deglutition and respiration are next to impossible. The entire neck externally is greatly swollen, and there is general asthenia.

Other varieties of swelling will be studied under the head of Elevations.

Enlargements of the tonsils include those dependent upon acute tonsillitis in its several varieties, and chronic tonsillar hypertrophy. In *acute catarrhal tonsillitis* the swelling is usually but slight, and may involve one or both glands. The adjacent portions of the soft palate and pharynx participate in the inflammation. A clear whitish exudation may be seen in the tonsillar crypts. In *herpetic tonsillitis* the swelling is greater than in the catarrhal variety, and the inflammation is apt to involve the fauces and the entire pharyngeal walls. Its special feature, however, is the presence of small vesicles on the tonsils and neighboring structures. (*Vide infra.*) *Acute ulcerative tonsillitis* has as its distinguishing feature the ulceration, which will be described later. The greatest swelling is observed in suppurative tonsillitis, also known as quinsy, and peritonsillar abscess. This affection seems to be especially liable to attack persons who already have enlarged tonsils, and is dependent upon infection plus a defective general condition. The enlarged tonsil is associated with œdematous swelling of the soft palate and uvula. Usually there is an early discharge of pus, which is rarely large in amount when the changes are limited to the tonsils themselves. Sometimes, however, there is a sloughing of a portion of the gland, leaving a large ulcer, which heals but slowly. This affection is usually attended by the constitutional phenomena of suppurative inflammations. In *peritonsillar abscess* the general condition is more severe, and the inflammatory swelling more extended, with glandular involvement. Fluctuation is generally made out beween the upper and middle two-thirds of the anterior pillar of the fauces. When it discharges spontaneously, the pus ordinarily escapes at the opening between the tonsil and the anterior pillar.

Tonsillar hypertrophy shows itself as enlargement of these glands projecting towards the middle line ; in extreme cases the opposite glands almost meet. They rarely, however, interfere seriously with respiration, unless associated with other lesions, as adenoid vegetations, to which, indeed, most of the ill-effects hitherto attributed to this condition are in reality due. Hypertrophied tonsils are especially liable to occur in children of scrofulous constitution, and their growth is favored by repeated attacks of acute inflammation about the throat.

Increase in the capacity of the pharynx results from congenital or acquired deformities, and from atrophy of the mucous and muscular structures. It is commonly known as pharyngocele, and may be congenital or acquired. In the latter case there is often more or less narrowing of the œsophagus. The dilatation may be diffuse, or it may be limited to a particular portion, under the latter circumstances constituting a pocket or diverticulum. The walls of this diverticulum are made up of mucous membrane and submucous connective tissue, and it is very apt to extend downwards and backwards between the œsophagus and vertebræ. Sometimes it forms a tumor which projects laterally in the neck, and which can be emptied of its contents by pressure. The causes of this deformity are imperfect mastication of food and hasty swallowing.

Enlargement of the pharynx and naso-pharynx is one of the physical signs of dry or atrophic pharyngitis. Inspection shows the mucous membrane to be thinned, dry, and apparently covered with a layer of thick varnish. Sometimes, too, there is a deposit of a green muco-pus, and even of crusts. This condition corresponds to atrophic nasal catarrh, presenting, as it does, all the physical evidences of that condition, and being always associated with it.

3. The condition of the uvula, soft palate, pharynx, and tonsils, as to movements, shape, secretions, etc.

The Uvula.—The deformities of this appendage are many, but are of little clinical importance with the exception of undue elongation, which is nearly always acquired, as a result of the irritation of frequent hawking or naso-pharyngeal catarrh. It may be œdematous, which condition arises from renal disease or local inflammatory changes, and debility. In cases of throat inflammation of high grade, it is nearly always greatly swollen from the engorgement, and œdematous. Enlargement of the uvula from any cause is liable to excite paroxysms of coughing and disturbances of respiration. Bloody extravasations into the uvula may occur in *peliosis rheumatica*, and in all diseased conditions capable of producing hæmorrhagic extravasations in other portions of the body.

The position of the uvula is sometimes of importance as suggesting disturbance of the motor function of the palate. Too much importance must not be attached to this, however, as is well illustrated in one of my cases in which it was for a time, at least, a moot question as to whether the dysphagia was paralytic or obstructive. The symptoms pointed to the latter cause, but the uvula deviated to one side; hence it was suggested that the trouble was of paralytic origin. Examination with the sound demonstrated the presence of an œsophageal obstruction, leaving us with the conclusion that the deviation of the uvula was but an accidental deformity.

The secretions and exudations of the uvula are the same as those observed on other portions of the throat.

Soft Palate.—The most frequently-observed malformation of the soft palate is a failure of union, usually at the median line, and generally associated with a similar condition of the hard palate. It is a congenital condition. Perforations as the result of ulcerations, especially of syphilitic origin, are also not uncommon.

The majority of ulcerations observed on the soft palate are manifestations of syphilis.

The palate is swollen more or less in all inflammatory affections of the throat. It is pushed forward in cases of post-pharyngeal tumors. Its motor-power is always impaired when the inflammatory infiltration is more than moderate—more so, however, in various paralytic affections, of which the following are the most important:

a. Post-diphtheritic paralysis.
b. Bulbar paralysis (glosso-labio-laryngeal paralysis).
c. Spinal accessory paralysis, usually produced by spinal caries or basilar meningitis.

Paralysis of the palate may be either bi- or unilateral. In bilateral paralysis the palate hangs lower than normal, and is not raised when the patient attempts to pronounce a long "ah." Attempts to swallow fluids are followed by regurgitation through the nose, and speech assumes what has been denominated a nasal quality. Pronunciation of sounds requiring the approximation of the soft palate to the posterior pharyngeal wall is impaired. For example, the patient says "bang" for "bag." In the case of unilateral paralysis the difficulty is to be recognized only by inspection, the trouble showing itself by limitation of the palatine movements to one side.

The palatine paralysis succeeding diphtheria is associated with loss of sensation. Irritation of the parts fails to arouse the usual reflex action, and electrical stimulation demonstrates the reaction of degeneration.

When paralysis of the palate is due to bulbar lesions it is associated with paralysis of the pharynx; hence its consideration as related to this affection will be postponed for a short period. There is very little to say concerning the subject as bearing on spinal accessory nerve disease beyond the fact that it is known that in some cases of this paralysis the muscles supplied by this nerve are atrophied.

Choreic movements of the soft palate have been described. The disorder is usually made evident first by a clicking sound audible to the bystander, and dependent upon approximation and separation of the tongue and palate. Two varieties of this spasm have been described. One is re-

referred to as voluntary or mimetic, and the other as involuntary or choreic. They are differentiated by reason of the fact that the latter are rhythmical in their succession, and the movements of the palate are visible on inspection of the throat, while they disappear in the mimetic variety.

Palatine spasm is said to be associated with nasal spasm. Such cases are very rare, and probably dependent upon associated lesions of the spinal accessory.

Paralysis of the pharynx is manifested clinically by failure of deglutition; food refuses to pass from the pharynx into the œsophagus. There is less difficulty in swallowing pulpy food than either fluids or solids. As it is usually associated with palatine palsy, fluids are forced out by way of the nose. In some cases particles of food enter the larynx and cause choking. Unilateral paralysis of the pharynx causes little or no functional difficulty. Pharyngeal paralysis may be symptomatic of nuclear disease, in which case other nerve nuclei are involved, as in glosso-labio-laryngeal palsy from meningeal disease and from disease of the bones of the skull. Occurring as one of the phenomena of post-diphtheritic paralysis, it is dependent upon a parenchymatous neuritis, and is associated with loss of power of the soft palate and of the larynx. These cases are always to be regarded with considerable concern.

Nuclear pharyngeal paralysis constitutes part of the clinical phenomena of bulbar paralysis. As already stated, the lips and tongue are also paralyzed, the loss of power starting usually in the tongue. Deglutition is rendered difficult from manifold causes. In the first place, the glossal palsy makes it difficult to transfer the bolus of food from the mouth to the pharynx; the disturbed motor function of the palate causes fluids to regurgitate through the nose; the weakness of the pharyngeal muscles (the paralysis complete only in the terminal stage) interferes with the onward passage of the food; and the occasionally associated laryngeal palsy permits the escape of particles of food into the larynx.

Spasm of the pharynx may be observed in

a. Hydrophobia.
b. Hysteria.

(*a*) **Hydrophobia.**—The spasms of the pharynx in this disease are preceded for a few days by an uncomfortable sensation about the throat. Then come attacks of choking and difficulty in swallowing liquids. Next, attempts at swallowing bring about spasms of the pharynx, owing to hyperæsthesia of the parts. The phenomena increase in severity and extend to other parts, notably to the muscles of respiration. The increasing general hyperæsthesia leads to their renewal on the slightest external irritation of any kind, as a draught of air or a visual impression. Finally the spasms become general, as shown by the widely-distributed muscular rigidity, opisthotonos, etc.

(*b*) **Hysterical spasm of the pharynx** is nearly always associated with other evidences of that disease. It constitutes the basis for that well-known and frequently-encountered symptom, the globus hystericus. Exceptionally well-marked spasm of hysterical origin may occur as an isolated phenomenon, as in the instance quoted by Gowers—that of a man who could not swallow in the presence of others.

4. **Vessels of the Pharynx.**—Ordinarily, the minute blood-vessels of the pharynx are not visible as such. In cases of chronic pharyngeal inflammation, however, they may become dilated and tortuous. This remark applies to both arteriole and venules, dilated venules being observed the more frequently.

5. **Exudations on the Palate, Tonsils, and Pharynx.**—Here we have a most important subject from a clinical standpoint, for a lack of its proper understanding has led to many erroneous diagnoses of diphtheria. The exudates which may appear in the throat consist of

a. Mucus.
b. Pus.
c. False membrane.
d. Blood.

(*a*) **Mucus.**—Increased mucus-production is present in the early stages of all pharyngeal and tonsillar inflammations. In the late stages of chronic catarrh the mucous membrane becomes unnaturally dry and devoid of mucus, because of atrophy of the glandular tissue. Reference has already been made to an essential atrophic catarrh which is of this nature from the beginning, and is secondary to a similar process in the nasal cavities. The undue patency of the nasal passages permits a too free and forcible ingress of air into the pharynx, as a result of which the natural secretions of the parts are dried rapidly, and thus promote the formation of crusts. At the same time, the nasal pathological process extends to the pharynx. This condition is claimed by some to be an important predisposing factor in the production of some cases of pulmonary tuberculosis. A removal of the dried pharyngeal secretions in this disease shows the mucous membrane beneath to be smooth, pale and colorless.

(*b*) **Pus** is found on the pharyngeal mucous membrane only when discharged from an abscess connecting with that tube. In all inflammations of the mucous membrane, when fully developed, the discharge is muco-purulent.

(*c*) **False Membranes.**—The subject of false membranes in the throat has a most important diagnostic significance, for it is lesions of this character which indicate the presence of diphtheria. At the same time, one must remember that every false membrane appearing in the pharynx or on the soft palate and tonsils is not due to diphtheria, *i.e.*, to the Klebs-Loeffler bacillus.

The true diphtheritic membrane may be situated on either the soft palate, uvula, pharynx or tonsils, and is adherent. When forcibly removed, it leaves a raw or bleeding surface beneath. The patches of membrane are raised slightly above the surrounding structures. At its outset it constitutes a grayish-white pellicle on the mucous membrane, which increases in size and thickness, until finally it presents an appearance which has been compared to white or grayish-white parchment. In its final stages it may be brownish, or even black. Bacteriological examination shows the Klebs-Loeffler bacillus, which is 2 to 3 μ long and 0.5 to 0.8 μ in width, straight or slightly curved. The pseudo-diphtheritic membrane resembles the true diphtheria very closely, and is recognizable only by bacteriological examination.

This seems to be the most convenient place to refer to those exudates on the tonsils which are so frequently mistaken for diphtheria. As a rule, they consist of accumulations of inspissated mucus in the crypts of the tonsils. When removed with a probe they are found not to be attached to the mucous membrane beneath, and consequently do not leave a raw, bleeding-surface. They are usually due to tonsillar inflammation, and are not infrequently observed in scarlatina. The frequency with which they are confounded with diphtheritic membrane has led to casting doubt on the diagnosis of diphtheria in all cases in which the deposit is limited to the tonsils. It must be borne in mind, however, that diphtheritic deposits may in their earlier stages, or even throughout the course of the disease, be limited to these parts; hence location must not be regarded as an infallible guide.

More confusing, probably, are the exudations covering pharyngeal ulcerations. Sometimes it is almost impossible to determine their nature by their objective appearance alone. In such a case one must look to the associated symptoms for help. In true diphtheria, the constitutional symptoms, *i.e.*, the toxæmia, are usually present. The fever is not apt to be severe, and glandular involvement and albuminuria are present.

(*d*) **Blood.**—While hæmorrhage may originate in the pharynx from a great variety of causes, it is important to remember that it is a very rare phenomenon, and that the majority of cases of blood-spitting are due to pulmonary tuberculosis. The causes giving rise to pharyngeal hæmorrhage may be tabulated as follows:

i. Alterations in the composition of the blood, as the anæmias, certain infectious diseases, and Bright's disease.
ii. Abscesses.
iii. Ulcerations, usually, however, lupoid and malignant.
iv. Post-nasal adenoids.
v. The rupture of enlarged veins in the pharynx, especially in gouty subjects and those having cirrhosis of the liver.
vi. Traumatism from foreign bodies.

6. **Ulcerations of the Pharynx and Tonsils.—Follicular ulcerations** are observed in acute follicular tonsillitis, and in many cases of chronic catarrhal inflammation. They are small and superficial, and are located on the posterior pharyngeal wall. In some cases they are preceded by the formation of small vesicles (herpetic pharyngitis), to the rupture of which they are due.

Deep ulcerations are occasionally observed in the course of scarlatina, diphtheria, typhoid fever, syphilis, cancer, and tuberculosis.

Syphilitic ulcers of the pharynx may occur in either the secondary or tertiary stage of the disease. In the former case they are for the most part superficial, painless, and usually situated on the posterior wall. They are not infrequently associated with mucous patches in the pharynx, and, more characteristically, in the mouth. In tertiary syphilis the ulcers are deep, and often serpiginous. On healing they leave dense, white cicatrices, which are generally regarded as diagnostic. Like the secondary ulcerations, they are painless, and they are not associated with glandular involvement. They may prove very destructive, involving, as they may, the entire soft palate and pharyngeal wall. There are times when it is impossible to distinguish them from tubercular or cancerous ulceration, excepting by the therapeutic test.

Tubercular ulceration of the pharynx is practically never a primary affection. Hence one finds a ready diagnostic help in the presence of late tuberculous lesions elsewhere, especially, however, in the lungs. They are generally grayish in color. Unlike the syphilitic ulcers, they are exceedingly painful, and are attended by glandular involvement. Microscopic examinations of scrapings from their surface will almost certainly discover the presence of the tubercle bacillus. They are irregular in outline, and present a grayish base. Occasionally, tubercular ulcerations may involve the tonsils in conjunction with similar lesions of the pharynx.

Malignant disease of the pharynx is nearly always secondary to malignant growths elsewhere, although primary cancer of the pharynx is occasionally observed. Usually the lesion starts in the neighborhood of the larynx. There is apt to be hæmorrhage as the initial symptom to direct the patient's attention to the state of his health. Then come pain, dyspnœa, dysphagia, and, in the case of epitheliomata, glandular involvement. The ulceration is secondary to the formation of the tumor, and it produces marked infiltration of the surrounding structures. Malignant ulcerations are noteworthy for their rapid progress. Whether occurring on the tonsils or in the pharynx, the lesion is progressive, spreading in both surface and depth, and the discharge from the same is sanious and offensive.

7. **Elevations, Including Tumors.**—The most frequently-observed elevations in the pharynx are those dependent upon enlargement of

the lymph follicles. Indeed, they are of such common occurrence that they may almost be regarded as a normal condition. They are especially numerous in persons of lymphatic constitution.

Enlargement of the mucous glands due to obstruction of their orifices and retention of secretions are common in many cases of chronic pharyngitis.

Vesicles arranged in similar form on the palate or pharynx are indicative of *herpes*.

Tumors of the pharynx include both benign and malignant. In either case they are rare. Their symptomatology depends largely upon their size and location. They are to be diagnosed only by their objective appearances.

8. **Subjective and Functional Symptoms in Pharyngeal and Tonsillar Diseases.**—These include:

a. Pain.
b. Sense of dryness.
c. Painful swallowing. (Dysphagia.)
d. Difficult breathing. (Dyspnœa.)

(*a*) **Pain.**—In the acute inflammations of the pharynx and tonsils the reason for the constant presence of pain is evident. It is noteworthy, however, that this pain is a source of more than ordinary suffering, because the functions of the pharynx necessitate frequent movement of the parts, thus adding to the irritation. In that not uncommon affection of the pharynx, muscular rheumatism, the pain is a prominent feature, while objective changes are slight or absent.

(*b*) **Sense of Dryness.**—This is observed in the initial stages of all acute inflammatory affections of the throat, and in many cases of chronic catarrh, especially the so-called atrophic and dry forms.

(*c*) The **dysphagia** may be due to either pain or obstruction; usually, however, to the former. It can be obstructive only in the presence of considerable inflammatory swelling or a tumor.

(*d*) **Dyspnœa** is rarely present in throat troubles, because it must be then due to obstruction. Its most frequent causes are tumor, abscess, phlegmonous pharyngitis, greatly enlarged tonsils.

(*e*) **Sensory Disturbances of the Pharynx.**—These are as follows:

i. Anæsthesia, which occurs in hysteria, diphtheria, and bulbar paralysis.
ii. Hyperæsthesia, which is an idiosyncrasy with some people, but it may occur pathologically in hysteria, mental affections, and hydrophobia.
iii. Paræsthesia, which is nearly always of hysterical origin. It may constitute one of the initial symptoms of malignant disease.

(i) **Anæsthesia of the pharynx,** aside from the cases in which it occurs as one of the phenomena of hysteria, is to be regarded as evidence of organic disease. It is to be recognized by manipulation of the parts with a probe. It is nearly always associated with paralysis, and hence is observed in the paralytic affections already mentioned,—most frequently, however, in connection with post-diphtheritic paralysis. It may also be due to lesions interfering with the function of the glosso-pharyngeal nerves, as tumors at the base of the skull or near the point of exit of the nerve from the skull.

(ii) **Hyperæsthesia of the pharynx** is the normal condition of many persons. In some cases it exists to such a degree that the slightest manipulation of the parts is capable of producing severe retching, and even vomiting. In many of these individuals the mental factor is a very important one, for the mere entrance of the probe into the mouth excites reflex contractions before the mucous membrane has been touched. These persons are for the most part highly neurotic; but the subjects of gout, phthisis and pharyngeal cancer may be similarly affected. In the latter it not infrequently constitutes the initial symptom of disease. The pharyngeal hyperæsthesia of hydrophobia is the cause of the spasms of the throat in that dread affection.

(iii) **Pharyngeal paræsthesiæ** are very common in practice, especially in chlorotic or anæmic young women and women at the climacteric. Although a neurotic habit favors their occurrence, it is by no means necessary to the incidence of the trouble. Their character is most varied, consisting, as they do, of vague general sensations, feelings of dryness or stuffiness, tickling, scraping, choking, as of a foreign body. The manner, too, in which the paræsthesiæ affect the patients is not always the same. In some they act as a mere inconvenience for which relief is sought; at the same time, the nervous system generally maintains a good balance. In others, however, the reverse condition obtains. The symptom becomes most obtrusive, and is never out of mind. In consequence there is general nervous instability, as exhibited by mental depression, more or less constant crying, insomnia, etc. Very often the hypochondriacal disposition is decidedly manifest, the patient imagining the existence of cancer, tuberculosis, or some equally serious disorder. Such cases demand the highest degree of medical judgment in their care; for, when it is certain that the trouble is purely neurotic, nothing is more important than encouraging assurance of the future. On the other hand, the physician must guard his own reputation for accuracy in view of the fact that many organic diseases have various paræsthesiæ as their initial symptoms; hence the importance of most painstaking physical examinations, to detect the same at the earliest possible moment.

The relation of these paræsthesiæ to the introduction of foreign bodies in the throat is a most practical subject, inasmuch as many of the cases find their origin in this accident. It is important to bear in mind that the power of localization of sensations in the throat is extremely defective; for the majority of sensations, no matter where their origin may be, are referred to the front part of the neck corresponding to the larynx and upper part of the trachea. If the physician is not aware of this fact, failing to find the suspected foreign body at the point indicated, he is liable to consider that it does not exist. On the other hand he may, by paying too much attention to the nervous symptoms, regarding them as positive evidence of a structural condition, intensify the morbid sensations.

Synopsis of the Symptoms and Clinical Course of Diseases of the Pharynx and Tonsils.

1. Acute Catarrhal Pharyngitis.

This disease has as its chief exciting cause exposure to cold. It may also originate in exposure to dust, hot air or chemical fumes, and to micro-organisms. Predisposition is an important factor in many instances. The latter is favored by bad hygienic surroundings, bad ventilation, excessive indulgence in alcohol and tobacco, and sedentary occupations. Persons of a rheumatic constitution are especially liable to be attacked.

The onset of the disease is usually sudden, being associated with high fever, and sometimes by general prostration and pains or aching over the body. Locally there is more or less pain in the throat, varying in severity, but aggravated during attempts at swallowing. The Eustachian tubes may participate in the inflammation, in which case the patient complains of pains shooting from the throat into the ears. Examination usually shows an increased redness of the mucous membrane of the pharynx, soft palate and tonsils, with slight puffiness and swelling. In the very beginning the mucous membrane is abnormally dry; in the fully-developed disease there is increased production of mucus. Externally, the cervical glands are sometimes enlarged. The diagnosis is readily made, excepting in children, in whom the above phenomena may be symptomatic of some constitutional disorder.

2. Acute Gouty Pharyngitis.

This occurs in individuals who have previously given evidence of the possession of a lithæmic or uric acid diathesis. Exposure to cold is the usual exciting cause. The onset of the disease is sudden, the patient first experiencing pain and fullness in the throat, aggravated during the act of swallowing. The throat also has a rigid, stiff feeling. When the disease is fully developed, the pain is disproportionately severe to the local appearances. The fauces and pharynx are of a bright-red color, the inflammation being of a decidedly patchy character. It is especially noticeable on the lateral pharyngeal walls. The uvula may be œdematous. As a rule, fever is but slight or absent.

3. Rheumatic Pharyngitis.

The objective appearances of rheumatic pharyngitis are either slight or absent, as one would expect from the nature of the affection, which is a disease of the pharyngeal muscles. Pain on swallowing is pronounced, and, moreover, is exceedingly resistant to treatment. Between the acute attacks, the patient complains of more or less sensitiveness of the throat.

4. Herpes of the Pharynx; Herpetic Pharyngitis.

Like most of the acute pharyngeal inflammations, herpetic pharyngitis seems to find its most important exciting cause in exposure to cold. Climatic influences are very important, for it is most frequently observed in damp climates, and during the spring and autumn. At times it presents well-marked clinical similarities to pharyngeal diphtheria, from which it frequently can only be differentiated by a bacteriological examination. This similarity is rendered all the more serious by the liability of this disease to occur during diphtheria epidemics.

If the case is seen in the beginning, the objective appearances are such as to place the diagnosis beyond question. Inspection of the pharynx then reveals the presence of numerous vesicles, which may appear in successive crops. These rupture in the course of twenty-four or forty-eight hours, leaving small, shallow ulcers, which have been compared to the aphthous ulcerations of the mouth. They are not infrequently so closely placed that they coalesce and become covered with a false membrane, and it is then that the disease is liable to be mistaken for diphtheria. The patient experiences considerable pain in the throat, especially on swallowing; also general prostration, and some fever. Herpetic pharyngitis offers a favorable prognosis, recovery ensuing within a few days, excepting in those cases in which one attack succeeds closely upon another.

5. Phlegmonous Pharyngitis.

Semon contends that this and erysipelas of the pharynx, gangrenous pharyngitis and acute inflammatory œdema are identical diseases, dependent upon varying degrees of virulence of the same pathological process, basing his opinion upon clinical and bacteriological evidence. However this may be, it is convenient to recognize them as separate affections and present the symptomatologies of each.

Phlegmonous pharyngitis occurs in persons whose constitutions have deteriorated under unhygienic surroundings, and after the infectious fevers. The inflammation involves the deeper structures of the pharynx, even to the deep fascia. It may take on a sloughing character, with resulting hæmorrhage. The tonsils are always highly inflamed, although the deep tissues of these glands need not necessarily be involved. The onset of the disease is sudden, and is associated with rigors, high fever, headache, and profound prostration. The throat is filled with offensive mucus. Pain is severe. The prognosis is highly unfavorable. This disease is not infrequently associated with erysipelas of the face.

6. Gangrenous Pharyngitis.

This disease is regarded by Trousseau as the pharyngeal analogue of noma. It is ushered in by high fever and severe pain in the throat. Inspection shows a severe inflammation of the pharynx, the parts assuming a dusky color, soon followed by the formation of an eschar. Prostration is profound from the beginning. The temperature may fall below the normal, while the pulse is slow but weak. The general integument may display petechiæ, on account of the profound alterations in the blood. The breath is horribly offensive. The larynx and œsophagus may be invaded. Finally, there is fœtid diarrhœa. If a favorable result ensues, repair is exceedingly slow. The prognosis is, however, almost invariably unfavorable, death taking place from exhaustion or pulmonary complications.

7. Retropharyngeal Abscess.

This condition has been sufficiently described. (*Vide* p. 164.)

8. Pharyngeal Tuberculosis.

Tuberculosis of the pharynx is practically always secondary to tuberculosis elsewhere. It should be remembered, however, that primary tuberculosis in this locality is a possibility. The primary focus of the tuberculosis in the case of pharyngeal lesions is usually found in the lungs or larynx. In many instances of tubercular laryngitis, the pharyngeal lesions are the result of direct extension from the epiglottis and larynx. Tuberculosis of the pharynx may be acute or chronic.

(*a*) **Acute pharyngeal tuberculosis** begins rather sharply with pain in the fauces, and other phenomena of acute pharyngeal inflammation. Indeed, should it not be suspected that the patient has a pulmonary lesion, the true nature of the illness may not be suspected. Inflammatory infiltration of the soft parts soon takes place, and this produces stiffness and a paretic condition of the soft palate, uvula, and pillars of the fauces. Thus it happens that food exhibits a tendency to enter the posterior nares during deglutition. In the course of a few days, several discrete, muddy-gray, slightly elevated miliary tubercles appear. These break down and form ulcers, which are sometimes so small as to escape recognition, excepting by careful inspection. In other cases they coalesce and form large ulcerations, irregular in outline, with grayish-white base and accumulation of caseating matter, and irregular "worm-eaten" edges. Pain is severe; indeed, it may render deglutition impossible. Other symptoms dependent upon the primary tubercular lesion are also present.

(*b*) **Chronic pharyngeal tuberculosis** occurs in conjunction with advanced pulmonary tuberculosis. The miliary granules are less prominent, and the ulcerations more so than in the acute form as above described. The ulcerations are indolent and less painful.

9. Pharyngeal Syphilis.

The pharynx may be the seat of primary, secondary or tertiary syphilitic lesions. Primary syphilis of the throat is rare and difficult of diagnosis, the tonsils being the usual location of the lesion. Chancres have been observed in the vicinity of the Eustachian tube through infection by the Eustachian catheter. They are manifested as indolent inflammatory nodules, which soon break down, to form ulcers with an indurated base. In most instances it is well-nigh impossible to discover the special characteristics of the primary lesion because of the widespread inflammation of the mucous membrane.

The secondary lesions appear at about the same time, after the primary sore, as do the constitutional manifestations, *i.e.*, the cutaneous eruptions. Moreover, they may be regarded as the mucous membrane prototypes of the latter. They include erythema, catarrhal inflammation, mucous patches, and ulcerations. Much stress has been laid upon the color of the erythema, it being generally described as dusky or dirty-red, interspersed with portions of mucous membrane of normal hue, thus giving the pharyngeal walls a characteristic mottled appearance. The lesions are always symmetrical. The *mucous patches* present the same features as do similar lesions of the mouth. They may occur on the soft or hard palate, the pharynx, or the tonsils. They are rarely observed on the posterior pharyngeal walls. Not infrequently, these patches result in superficial ulcerations.

The *tertiary lesions* do not, as a rule, appear until at least six or seven years after the primary infection. They may also be observed in hereditary syphilis, but rarely until after the patient has reached the fifteenth year of age. They include the formation of gummatous growths, which, if not properly treated, break down and form destructive ulcers. Cicatrization is followed by distortion of the parts.

10. Chronic Catarrhal Pharyngitis

Is characterized by its gradual onset. It is due, in the majority of cases, to repeated exposure to unfavorable climatic conditions, tainted atmospheres, excessive indulgence in tobacco and alcohol, improper vocalization, and nasal stenosis. The subjective symptoms are a source of great annoyance, and include burning, stuffy, pricking, tickling, and other sensations in the pharynx. There is a persistent accumulation of mucus in the pharynx, forcing the patient to make frequent attempts at clearing the throat by the acts of hemming and hawking. Inspection shows the mucous membrane of the pharynx to be more or less congested. The walls of the pharynx are studded with numerous small elevations, believed to be the result of glandular hypertrophy. The pharyngeal mucous membrane is covered with mucus. An irritable hacking cough is not unusual. The hearing is sometimes involved, owing to the extension of the inflammation to the Eustachian tube. Pain is rare.

11. Atrophic Pharyngeal Catarrh

Is claimed by some to be an advanced stage of the preceding affection, the mucous membrane of the pharynx undergoing atrophy of its glandular structure and presenting a dried appearance. Moure claims that it is an extension of an atrophic rhinitis, and that it tends to spread to the larynx, producing atrophic laryngitis. The subjective symptoms include local discomfort, forcing the patient to clear the throat at short intervals, thinning and dryness of the mucous membrane of the pharynx, the collection of crusts of inspissated mucus upon the pharyngeal walls, and an unusual roominess of the pharynx.

12. Follicular Pharyngitis

Is a chronic inflammation involving, chiefly, the glandular structure of the pharynx. It is also known as *clergyman's sore throat* and *granular pharyngitis.* It has as its predisposing causes impoverished constitution, anæmia, the neurotic temperament, the rheumatic and gouty diathesis, and habitual use of tobacco. As exciting causes, improper or excessive use of the voice and the taking of hot or highly-seasoned foods are the most important.

The main symptom, and the one upon which the diagnosis of the affection is based, is an enlargement or distention of the muciparous glands of the pharynx. In the beginning of the affection there is increased secretion. Later, the glands inflame. Then the secretion becomes lessened and altered in quality. The patient now complains chiefly of the dryness of the throat. The parts become tired from continued use of the voice, which may exhibit marked alterations from its normal qualities. The course of the disease is essentially chronic, made so not only by the obstinacy of the lesions, but also by the underlying dyscrasia and the personal habits of the patient.

13. Acute Catarrhal Tonsillitis

Is due mainly to exposure to cold, or to atmospheric changes, especially in rheumatic subjects. Its onset is rapid, and is attended by more or less fever in association with constitutional symptoms, as headache, backache, general pains, etc. Locally the patient complains of severe soreness of the throat, which is aggravated by the act of swallowing. The tonsils are red and swollen, but the swelling is limited to the mucous and submucous tissues, and hence it is not extreme. Occasionally, there is associated swelling of the submaxillary lymphatics. The disease runs a course of two or three days.

14. Acute Suppurative Tonsillitis or Quinsy

Is due to the same causes as the preceding, plus pyogenic infection. Its onset is rapid, and is attended by high fever and prostration, headache, and backache. As a rule, but one tonsil is involved, the other being but superficially inflamed. Inspection shows great tonsillar swelling, the affected gland presenting a peculiar redness, which to the experienced eye is characteristic. There is nearly always marked glandular involvement, and swelling of the peritonsillar tissues. Pain is intense; indeed, it prevents any attempt at deglutition. Severe pains may shoot from the throat into the ears. At times the constitutional disturbance is so great as to cause delirium. Suppuration is generally well advanced by the fifth day, at which time it is easy to discover fluctuation. All symptoms are relieved promptly after incision or spontaneous rupture of the abscess. Cases of quinsy should be watched carefully, lest rupture take place during sleep and lead to strangulation; but this is a rare accident. Tonsillar abscesses have been known to open into the carotid artery and cause serious hæmorrhages; but this, likewise, is a very unusual occurrence.

15. Acute Follicular Tonsillitis

Occurs in rheumatic and neurotic subjects. It is apparently dependent upon seasonal and climatic influences, and is probably feebly contagious. It is characterized by its rapid onset, with high fever, rapid pulse, general prostration, aching of the back and limbs, headache, etc. The throat is very painful, the pain being aggravated on swallowing. The tonsils are swollen, their crypts enlarged and studded with exudates composed of inspissated mucus. Such accumulations are readily removed, leaving an intact but inflamed mucous membrane beneath. Deposits are not seen on the palatine or pharyngeal mucous membrane. The cervical glands are usually enlarged. Follicular tonsillitis usually runs a course of three or four days, and is followed by more or less pronounced prostration. Albuminuria is sometimes present. This disease is very frequently confounded with diphtheria.

16. Acute Herpetic Tonsillitis

Presents the same general and subjective symptoms as other acute tonsillar affections. Objectively, there appear numerous small vesicles on the tonsils and pharyngeal walls. This disease has also been mistaken for diphtheria.

17. Enlargement of the Tonsils

Has been studied sufficiently when reviewing the various throat symptoms *seriatim*. (*Vide* p. 165.) It is only necessary to enforce upon the mind of the reader the frequency with which these lesions are associated with post-nasal adenoids. Indeed, it is often contended that many of the serious symptoms formerly alleged to be dependent upon tonsillar hypertrophy are really the result of the associated condition.

18. Diphtheria.

Diphtheria is always dependent upon infection with the Klebs-Loeffler bacillus. This statement is made in full knowledge that cases occur without an apparent exposure to infection ; nevertheless, facts demonstrate that such infection must have taken place, and that it is possible for persons to be unwittingly exposed to it. A predisposition is, of course, necessary ; such predisposition consisting of constitutional weakness, or local changes favoring the growth of the diphtheria bacillus when deposited upon the throat.

The symptoms of diphtheria are constitutional and local. Both of these classes of clinical phenomena are asserted in the beginning of the affection. The *constitutional symptoms* include fever, rapid and usually weak pulse, and prostration: The intensity of the *fever* varies greatly in different cases. In some it is quite high ; in others, perhaps the great majority, it is but moderate. The *pulse* is always rapid ; more so, indeed, than the intensity of the fever would warrant. Its loss of strength depends in great measure upon the severity of the constitutional infection. The onset of prostration varies greatly in time. In most instances it is a prominent feature from the beginning. In some few cases it is not manifested until late ; indeed, the maintenance of the usual strength of the patient during the early days of the illness may lead to neglect of proper attention.

The diagnosis of the affection is based upon the local condition of the throat. The characteristic symptom is the appearance of a false membrane, which is first manifested as a small grayish or dirty white spot, which in the course of a few hours enlarges even to the extent of covering the entire fauces, soft palate and pharynx. This membrane may be thin or thick ; it may be attached closely or loosely to the subjacent mucous membrane. In any event, its detachment leaves a more or less raw surface.

With the progress of the case, its color may change to a brownish hue,—always a serious sign. The surrounding mucous membrane is highly inflamed and swollen. Fœtor—sometimes extreme—is commonly present. There is generally infection of the lymphatic gland beneath the jaws. As a rule, pain in the throat, aggravated on swallowing, is severe.

The membrane does not always remain limited to the throat. It may extend into the nasal cavities above or into the larynx below. In either case the gravity of the prognosis is much increased. When involving the nose, the lumen of the nasal passages may be obstructed. At the same time, there is frequently a profuse excoriating coryza. Discharge from the nostrils is, however, frequently absent; but the physician should not depend upon the non-appearance of this symptom as an assurance that the nasal cavities are not involved, and neglect to make regular examinations of the same.

More serious is the extension of the membrane to the larynx,—a complication which is indicated by obstructive dyspnœa and the characteristic brassy or croupy cough of laryngeal disease.

Less common extensions of the membrane are to the conjunctiva, œsophagus, bronchi, and ears.

In nearly all cases, examination of the urine will discover the presence of albuminuria. This symptom usually makes its appearance about the second or third day. Microscopically, hyaline casts may be present. In some cases there is an actual nephritis, in which case the urine becomes scanty, and there may be œdema, and ultimately uræmia may set in. The urine in these cases contains granular casts and red blood cells.

The most important of the complications of diphtheria is the toxic neuritis so frequently observed during convalescence. This gives rise to various types of paralysis. In the majority of cases it is the eyes, throat and heart that are involved, producing paralysis of accommodation, paralytic dysphagia and heart failure, respectively. In the latter we have the usual cause of sudden death in diphtheritic patients. I have said sudden death; and yet I believe that, with careful attention to the condition of the patient, the incidence of cardiac paralysis may be prophesied some little time in advance of its occurrence.

The multiple neuritis, which is also of common occurrence, produces paralysis more or less widespread, according to the number of nerves involved. For further particulars concerning this affection, the reader is referred to the pages devoted to the study of neuritis in general.

Ordinarily, the diagnosis of diphtheria is not a difficult matter. Cases will arise, however, in which the clinical phenomena are not sufficient, and recourse must be had to a bacteriological examination at the hands of the expert. But here one should not permit his judgment to run wild, for it is admitted that the Klebs-Loeffler bacillus may be found in perfectly normal throats without the case being one of diphtheria. They have simply found lodgment in non-fertile soil. And, again, it may happen that carelessness in examination technique may fail to discover them when present. While not decrying the importance of such examinations, for no one appreciates their value more than does the author, the physician should accept them only in conjunction with the local symptoms of the disease. In doubtful cases they should always be permitted to decide the nature of the case. Theoretically, of course, the bacteriological examination should be the sole guide; but in clinical medicine, theories are only of use as working-hypotheses.

Diphtheria is always a serious disorder. Its prognosis is decidedly less unfavorable since the discovery of antitoxin. Indeed, I believe, with an early diagnosis and the prompt institution of the antitoxin treatment, the mortality should be reduced to practically *nil*.

The Œsophagus.

Anatomical and Physiological Considerations. — The œsophagus is a tube from three-quarters of an inch to a scant inch in diameter, the function of which is the transmission of food from the pharynx to the stomach. It starts opposite the cricoid cartilage of the larynx, *i.e.*, on a level with the sixth cervical vertebra. In its downward course it lies to the left of the spinal column until it reaches the level of the fifth or sixth dorsal vertebra, at which point it crosses to the right. It is crossed by the left bronchus opposite the fourth or fifth dorsal vertebra. Before reaching the diaphragm, however, it recrosses to the left, and finally enters the cardiac extremity of the stomach at the level of the tenth or eleventh dorsal vertebra. In the neck it is in relation in front and at the sides with the thyroid gland, and at the sides with the recurrent laryngeal nerves. Above the point at which it is crossed by the left bronchus the œsophagus is in relation with the trachea in front and the recurrent laryngeal nerves on either side; below this crossing it bears close anatomical relations with the bronchial glands, the aorta, pleuræ and pericardium.

Some Important Measurements.—The average length of the œsophagus in the adult is from nine to ten inches; from the edges of the incisor teeth to the cardiac orifice ranges from fifteen to sixteen inches; from the edge of the incisor teeth to the point of crossing by the left bronchus is eight inches.

Methods for Examination of the Œsophagus.—These include

1. The sound.
2. Auscultation.
3. Inspection.
4. Palpation.

1. **The Sound or Tube.**—Examination of the œsophagus by sounding is indicated only when obstructive disease of that tube, as stricture or sacculation, is suspected. The best instruments for the purpose are specially-constructed bougies, oval in cross-section, and ranging in diameter from one-eighth to one-half inch. They must be perfectly flexible. If carelessly used, the sound is not devoid of danger; hence certain precautions must always be adopted. The possibility of aortic aneurysm being the cause of the suspected obstruction must always be entertained, and, if present, sounding should not be practised. In the first place, it will give no information that we do not already possess. If the symptoms suggest œsophageal stenosis, and if we find the physical signs and the symptoms of aortic aneurysm, it would seem that no further steps are necessary, in view of the possible dangers of sounding in these cases. The danger referred to lies in the fact that the long-continued pressure of the aneurysm on the œsophageal walls leads to their gradual thinning, and finally spontaneous rupture, with fatal hæmorrhage. Clinically, one can never recognize the degree of alteration in the œsophageal and aneurysmal walls; hence the wisdom of refraining from a diagnostic procedure which brings with it an element of danger without giving commensurate information and suggestions for treatment.

The œsophageal sound should not be used on persons who have recently vomited blood, and this whether suspected hæmorrhage originates in the œsophagus or in the stomach. In either case, the manipulation of the instrument or the reflex retching and vomiting may renew the bleeding.

Manner of Passing the Sound.—It is well to begin with one of the smaller sizes; if this is passed readily, then a larger one may be introduced. The sound or tube should be moistened with water or milk to act as a lubricant; grease of any kind is entirely unnecessary. The patient should be in a sitting posture. Ordinarily it is best to have the head thrown backwards very slightly, the tongue being moderately protruded. At the same time, it is well to remember that in some cases the introduction of the instrument is favored by bending the head slightly forward; in other words, the position of the head is largely a matter of circumstances. The tube is passed over the dorsum of the tongue and into the pharynx. Then the patient is requested to swallow, and as he attempts to do so the tube is pushed onward, with but slight force. Some little difficulty may be encountered at the œsophageal entrance because of the retching, but this is overcome with the exercise of a little patience. The fingers need never be introduced into the throat to direct the instrument, as it can go in but one direction, entrance into the larynx being impossible unless there are anæsthesia of the parts and paralysis of the epiglottis. Under such circumstances, the entrance of the sound into the larynx will produce severe dyspnœa. No difficulty is ever encountered in passing the sound after it has entered an œsophagus of normal calibre; after it has passed a distance of sixteen inches it is at the cardiac orifice. Any obstruction met with is to be regarded as pathological.

When passing the sound, pay attention to the following:

a. The presence or absence of pain; normally, there should be discomfort only.

b. Obstructions which may be either

  (i) Functional, occurring for the most part in hysterical subjects.

  (ii) Organic, which may come from changes in the œsophageal walls, as from malignant growths (rare); from cicatricial narrowing, following burns, as from swallowing corrosive alkalies; from pressure outside the œsophagus, as from aortic aneurysm, malignant tumors, enlarged glands; and from causes within the œsophagus, as a foreign body lodged therein.

c. Diverticula.

d. Freedom of movement of the tube; if too free, dilatation is suggested.

e. The character of the discharges on the tube after it has been withdrawn. Normally, there should be an abundance of thick, glairy mucus; but pathologically there may be blood, muco-pus or pus, indicating abrasions, ulcerations or abscess.

Functional obstruction of the œsophagus is due to spasm, and is known as *œsophagismus*. It is dependent almost entirely upon a neurotic constitution, and is especially observed in cases of hysteria in women and hypochondriasis in men. Brinton and Mackenzie have called attention to cases arising from gout. A most important class of cases is those in which the spasm occurs in persons who have been bitten by dogs, and who, in consequence, live in constant fear of hydrophobia. The mind finally becomes morbid, and spurious hydrophobia results. Prominent physicians have claimed that all hydrophobia is of this character; but the preponderance of evidence at the present time favors the view that it is an actual disease. Œsophagismus is unlikely to exist without the association of other nervous symptoms to explain its nature.

Hysterical œsophagismus is largely dependent upon idea; certain foods or certain circumstances make attacks possible; also direct attention to the ailment.

Organic obstruction is recognized by the symptoms of dysphagia, which are persistent, and by the detection of narrowing of the œsophageal calibre by the sound. The special character of the obstruction is to be determined by the history of the case and the associated signs and symptoms. Its situation is determined by measuring on the sound the distance at which the obstruction is encountered, and its calibre by the size of the instrument which can be passed.

Congenital œsophageal atresia is one variety. Examination demonstrates that the upper segment of the œsophagus ends somewhat above the level of the bifurcation of the trachea. Infants having this deformity die of starvation a few days after birth.

The most frequently-observed cases of obstruction are those depending upon cicatricial contraction after the ingestion of corrosive fluids, especially lye. Most of these accidents occur in children who have taken the "stuff" accidentally. The nature of the obstruction is usually determined by the history of the case, although its situation in the upper portion of the œsophagus is suggestive when such history is wanting.

In **cancer of the œsophagus** the symptoms of stenosis are of slow onset. Pain is present, as in all malignant affections, but is not apt to be severe. The passage of the tube may bring away particles of tissue, the microscopic examination of which settles the diagnosis. Pressure symptoms are not uncommon, and include cough from irritation of the recurrent laryngeal and pneumogastric nerves and dyspnœa from tracheal obstruction. Sometimes the ulceration of the tumor is quite extensive and opens into the trachea or a bronchus, leading to a variety of serious symptoms, due to entrance of food and discharges into the air-passages. Perforation is characterized by sudden onset of dyspnœa. The patient is nearly always advanced in years, and undergoes rapid emaciation disproportionate to the degree of stenosis. There may be expectoration of frothy mucus with blood-streaks; sometimes particles of cancerous tissue. It is said that in œsophageal cancer there is nearly always atrophy of the stomach. Sarcoma of the œsophagus is very rare; it is not to be diagnosed during life.

The diagnosis of **obstruction from œsophageal abscess** depends very largely upon the history of the case, unless the lesion happens to be high up and accessible to palpation. There must be a history of causes of suppuration—in this case acute œsophagitis or the introduction of a foreign body which has lacerated the walls, or the presence of vertebral caries. We expect the presence of rigors and fevers as evidence of the presence of a suppurative process.

Obstruction from aneurysm of the aorta is rarely sufficient to produce a high degree of dysphagia; certainly, fluids are never interfered with in their passage. The diagnosis is based upon the physical signs of aneurysm and a syphilitic history, or evidences of arterial degeneration.

Some slight obstruction may result from **enlargement or dilatation of the left auricle** or **pericardial effusion,** but it is not likely to be evident to passage of the tube. The patient expresses himself as conscious of a discomfort when food or liquid passes the point of narrowing. In such cases the diagnosis is probably made before any difficulty of deglutition is experienced.

Obstruction from **pressure of mediastinal glands** occurs in conjunction with malignant disease, Hodgkin's disease, tuberculosis, and syphilis.

Obstruction in the neck dependent upon pressure from without is ordinarily of easy recognition, though cases will occasionally present themselves in which great difficulties obtain.

Obstruction from foreign body may be due to the body itself, and to reflex spasm excited by its presence. Emphysema and excessive secretion of ropy mucus are important suggestive symptoms.

Diverticula of the œsophagus are to be suspected when the tube passes readily one time and meets with an impassable obstruction another. Palpation will usually discover a tumor on one side of the neck, becoming larger after eating. Compression empties it of its contents. The mode of origin of these pouches has afforded considerable discussion. Each view, probably, is applicable to certain cases. One claims that slight sacculations exist at birth, and increase in size as time goes on; another claims that, as the result of traumatism, there is a hernia of the mucus through the muscular coat of the œsophagus; and still a third provides for dilatation from traction of cicatricial tissue, which are hence known as traction diverticula. The usual situation for such pouches is near the bifurcation of the trachea, and are dependent upon adhesions between inflamed bronchial glands and the œsophagus. They give rise to no symptoms unless ulceration takes place.

Dilatation of the œsophagus may be either primary or secondary. In primary dilatation the entire length of the tube is involved, although the changes are especially prominent at the middle-third. In some cases the œsophagus is lengthened, and hence is made tortuous. Such a state of affairs will make the passage of the sound difficult, instead of abnormally free. The symptoms are regurgitation of food some hours after eating, neutral or alkaline in reaction, sensation of distention and burning pain along the œsophagus, and offensive breath. The etiology of the affection is not understood. Mackenzie has suggested that it is due to a congenital or acquired atony of the œsophageal walls. Retention of food is found in the secondary cases by the stricture.

A study of the **discharges from the œsophagus** need not be limited to those found on the tube after its withdrawal. The most important are mucus and blood. As already stated, it is a normal condition to find the sound covered with a glairy, clear mucus. In certain diseases, however, notably acute and chronic œsophagitis and cancer, this viscid mucus is produced in large quantity. It then wells up into the pharynx, whence it is expectorated by the act of hawking.

Hæmorrhage from the œsophagus is usually due to the rupture of aortic aneurysm, or to varicose veins about the cardiac orifice. In the former case the hæmorrhage is slight at first, but entirely resists treatment. It finally becomes profuse, and ends fatally. Sometimes the initial rupture is large, in which case the hæmorrhage is profuse from the first, and rapidly fatal. Hæmorrhage from rupture of varicose veins at the cardiac extremity is liable to occur in association with cirrhosis of the liver and other conditions, producing portal obstruction. Œsophageal hæmorrhage is not always easy of discovery. When the blood is discharged by way of the mouth, it will be found that it wells up into the pharynx, and is then expelled by clearing the throat. It is found to be bright in color and alkaline in reaction. Sometimes, however, the blood is swallowed, and, when the hæmorrhage is profuse, may so stimulate peristalsis as to appear by way of the rectum. Such cases call for most painstaking examination for their recognition. Generally their true nature escapes detection because the physical condition of the patient is too bad to permit thorough investigation.

The presence of a small quantity of blood on the tube indicates that there is an *ulceration*, the nature of which must be determined by attendant circumstances. The same remarks hold true respecting the presence of pus.

2. **Auscultation of the Œsophagus.**—Profitable auscultation of the œsophagus in the diagnosis of its diseases, especially stenosis, can be had only when the practitioner is conversant with the sounds attendant upon deglutition in health. These consist of a peculiar gurgling sound or series of sounds during the act of swallowing, heard best on the left side of the neck; a similar though fainter sound is audible over the left side of the upper six dorsal vertebræ. The normal sound is sharp and sudden, and ceases suddenly.

Inasmuch as the simultaneous entrance of air and liquid into the pharynx produces sounds which interfere with the true sounds made by the œsophagus, it is wise to direct the patient to drink continuously but slowly. It is also important to observe the time occupied by the deglutition-murmurs, and the periods at which they occur in relation to the beginning of the attempts at swallowing. For this reason it is a good plan to have one finger over the hyoid bone while listening with the stethoscope. The elevation of the hyoid indicates the exact second when swallowing commenced.

In cases of œsophageal stenosis, the deglutition-murmurs are either absent, weak, or delayed.

3 and 4. **Inspection and Palpation.**—These are limited, in their clinical application to disease of the œsophagus, to cases in which the lesion is situated in the neck. It is true that an instrument designed for the inspection of the œsophagus throughout its entire length has been designed, but its use requires such a high degree of technical skill that it is practical for specialists only. In cases of diverticula a tumor may present itself on the side of the neck, usually the left. It fills up after eating, and may

be emptied of its contents by compression, the evacuation being accompanied by splashing or rumbling sounds. An enlargement behind the trachea is readily palpable in cases of œsophageal abscess. Palpation is also used for the recognition of a symptom too seldom ascribed to œsophageal disease, namely, subcutaneous emphysema, which, when present, will give rise to sense of crepitation and swelling.

The **subjective symptoms** which direct attention to the œsophagus as the cause of the illness and suggest the propriety of a physical examination of this structure are:

a. Pain.

b. Difficult deglutition, or dysphagia.

(*a*) **Pain** is observed only in acute and chronic œsophagitis and malignant disease. It is especially severe in acute inflammation. It is experienced mostly in the neck, and in the back between the shoulders. It is aggravated greatly by attempts at swallowing; indeed, it may make deglutition impossible. The absence of pain following the ingestion of caustic fluids suggests that the chemical action has produced entire destruction of tissue.

External pain, described by the patient as stiffness of the neck, may be associated with œsophageal inflammations.

(*b*) **Difficult Deglutition.**—This may be dependent upon

(i) *Stenosis* from any of the causes already enumerated. (*Vide* page 184.)

(ii) *From the severe pain* accompanying the act, as in acute œsophagitis.

(iii) *Paralysis of the œsophagus.*

The dysphagia of stenosis is characterized by its gradual onset, and the fact that the patient is inconvenienced first by solid food, which must be reduced to a finer and finer subdivision before it can be swallowed; ultimately only liquids can be forced into the stomach, and in some instances the stenosis may progress to such a point that even these cannot pass the stricture. In these particulars the dysphagia of obstructive disease is characteristic.

(iii) **Paralysis of the Œsophagus.**—When dysphagia is dependent upon paralysis of the œsophagus, there is always associated loss of power in other parts, *e.g.*, the larynx and pharynx. The tube passes readily. Deglutition is difficult, but there is no regurgitation. As a rule, the trouble comes on very gradually. The conditions in which it occurs are cerebral hæmorrhage, bulbar paralysis, brain tumor, and general paralysis of the insane.

Cough, when caused by œsophageal disease, may be attributed to pressure upon the left bronchus or upon the recurrent laryngeal or pneumogastric nerves. The lesion, in such cases, is usually carcinoma.

The Stomach.

The important anatomical details of the stomach, from a clinical standpoint, relate to its size and position. The stomach is to be regarded as a dilated portion of the digestive tract connecting the œsophagus above with the small intestines below. It occupies the upper portion of the abdominal cavity in the region known as the epigastrium, three-fourths of the organ being to the left of the median line and one-fourth to the right. The cardiac extremity lies somewhat to the left and in front of the twelfth dorsal vertebra; it is opposite anteriorly to the sternal extremity of the seventh costal cartilage. Its deep situation makes it inaccessible to palpation.

The smaller curvature of the stomach, starting at the cardia, runs vertically downwards until, opposite the body of the first lumbar vertebra, it crosses to the right, and, curving upwards, ends in the pylorus.

As external landmarks of the positions of the gastric orifices, we have the ensiform and costal cartilages. The cardiac orifice is situated in the left parasternal line, somewhat above the ensiform cartilage; the pylorus level with the tip of the ensiform cartilage, and below the point of union of the right seventh and eighth costal cartilages. It is rather freely movable, passing downwards and to the right when the stomach is distended.

The lesser curvature and the pylorus are not accessible to palpation because of their position beneath the liver.

The greater curvature of the stomach starts at the cardiac orifice to the left, then curves downwards, and then to the right, crossing the median line to the pylorus. Its position varies greatly, according to the degree of distention of the stomach. In the average healthy individual it will be found to cross the median line at about two finger-breadths above the umbilicus. When distended it may reach to the latter point.

Dimensions of the Stomach.—The dimensions of the distended stomach have been stated by Sappey as follows: Between fundus and pylorus, ten to twelve inches; between lesser and greater curvature, four to five inches; between anterior and posterior walls, three and one-half inches; between cardia and pylorus, three to six inches.

The symptoms of disease of the stomach may be divided into three classes:

a. Those related to perverted function.
b. Those dependent upon the pathological process, and which are practically the same for that particular process, no matter in what portion of the body it may occur.
c. Constitutional symptoms, mostly arising from malnutrition.

(*a*) The **symptoms dependent upon perverted function** may be dependent upon

(i) Disturbed secretion.
(ii) Disturbed motor-power.
(iii) Disturbed power of absorption.

(i) The *secretion of the stomach* (the gastric juice) may be increased or decreased in quantity or altered in quality.

(ii) The *motor-power of the stomach* may be diminished (atony), or the organ may be unduly irritable, giving rise to vomiting or retching.

(iii) *Disturbed power of absorption* is dependent upon disease of the absorbing structures.

The **investigation of gastric disorders** demands systematic inquiry into the history of the case, and careful physical examination. When taking the history of the conditions, especial attention should be paid to eliciting the following:

1. The mode of onset, duration, and the alleged causes of the illness.
2. The presence or absence of pain; its character, its time of onset, its relation to the taking of food, and its association with other symptoms.
3. The condition of the bowels.
4. Degree of bodily emaciation.
5. The appetite, and the character of food taken.
6. The presence or absence of thirst.
7. The power of deglutition.
8. The presence or absence of flatulence.
9. The rapidity of progress of disease.
10. The condition of other organs, especially of the heart and lungs.

Methods for Examination of the Stomach. — For physical examination of the condition of the stomach we resort to the following procedures:

1. Inspection.
2. Palpation.
3. Auscultation.
4. Percussion.
5. Chemical analysis of the gastric contents.

1. **Inspection.**—This applies to the patient in general and the epigastric region in particular. The general appearance of the sufferer is suggestive of certain conditions, and enables us to exclude certain others. Thus, an appearance of general malnutrition or emaciation suggests some serious disease, *e.g.*, cancer. At the same time, it should be remembered that any organic disease of the stomach may bring about the same result, in time, by reason of the diminished amount of food taken. On the other hand, in gastric neuroses, no matter how great the discomfort of the patient

or how long continued, the general health, aside from the nervous system, maintains a comparatively good standard.

It is only in exceptional instances that much information is obtainable by inspection of the epigastrium. When the walls of the abdomen are unusually thin, it may be possible to note the exact outlines of the stomach. If that organ is dilated, then the data are made still more evident.

If inspection is unsatisfactory in its results, the stomach may be distended artificially, to make its shape, size and position more evident. For this purpose two methods are at our disposal. One is pumping air into the stomach by means of an especially-constructed apparatus. This method has the advantage of regulating the amount of distention to a nicety, and enables the operator to remove it in an instant should the patient become oppressed. It labors under the disadvantage of inconvenience. The other method consists of administering two teaspoonfuls of tartaric acid dissolved in water, followed in a few seconds by the same quantity of bicarbonate of soda in solution. Carbonic acid gas is evolved and distends the stomach, thus enabling the physician to outline the organ by inspection, palpation and percussion. If the rapidly-evolved gas produces oppression, as it sometimes does, it may be removed rapidly by passing the stomach tube. Occasionally inflation of the stomach is useless because of undue patency of the pylorus. In this case, the gas escapes into the intestines as fast as it is formed.

When the stomach is thus distended and the abdominal walls are sufficiently thin, the greater curvature becomes visible somewhat above the umbilicus; occasionally it may be as low as that point.

Local prominences are sometimes observable in case of tumors, especially when the latter are situated on the anterior wall of the stomach.

Sometimes inspection discovers peristaltic waves passing from left to right over the upper portion of the abdomen. These indicate the presence of the condition known as "peristaltic restlessness," and constitute one of the neuroses of the stomach. They are sometimes observed in connection with pyloric obstruction.

2. **Palpation.**—In palpation we have the most useful means of physical examination of the stomach. To secure the best possible results, care in its practice must be observed. First the patient must be made perfectly easy and comfortable in a recumbent position. The physician will find that he can work better standing to the right of his patient. Palpation then should be carried out systematically, investigating carefully every portion of the abdomen. At first it should be light, and with the fingers only; later, as the abdominal walls become thoroughly relaxed, it may be made deeper, and with the whole hand, if necessary. If there is a tendency to rigidity of the abdominal walls, the attention of the patient should be diverted from the examination, or he should be directed to breathe deeply

with the mouth open. When investigating the condition of an organ, the left hand should be used to push the viscus under investigation towards the right hand, which is the one seeking the information. If the patient does not relax thoroughly despite the most careful efforts to that end, and if the information obtained is not satisfactory, then the patient should be anæsthetized, and the examiner given every possible advantage.

When palpating, endeavor to obtain information as to the following points:

a. The size of the various abdominal viscera.
b. The presence or absence of abnormal growths.
c. The presence or absence of pulsations.
d. The presence or absence of sensitive points.

When sensitive points are found, a mental note of the organs or parts beneath such points should be made. Such tenderness is found in acute and chronic inflammations of the stomach, ulcer and cancer. It commonly serves as a basis for the diagnosis of ulcer, of which it is diagnostic only when permanent and strictly localized.

Attention should also be paid to the relation between abdominal rigidity and localized sensitiveness.

An increased resistance over the epigastrium is occasionally observed apart from any rigidity of the abdominal walls. This may be produced by hypertrophy of the muscular coat of the stomach, which may exist in association with distention of that organ, with *disseminated cancer* of the gastric walls, or with *fibroid stomach*, in which condition the mucous membrane and walls of the organ undergo atrophy and formation of large amounts of connective tissue. (Fibrosis.) This increased resistance or induration is observed more readily along the line of the greater curvature of the stomach, especially to the right of the median line. Very exceptionally, when the abdominal walls are thin, there may be felt a localized induration, due to the infiltration surrounding a gastric ulcer.

It is important to bear in mind that, because a tumor is not discoverable, it does not necessarily follow that one does not exist. It has been stated already that tumors about the cardia are not palpable because of the deep position of that part, while those of the pylorus not infrequently escape detection because of their position beneath the right lobe of the liver.

Tumor of the pylorus, when palpable, may be discovered between the ensiform cartilage and the umbilicus, but to the right of the median line. When not adherent to adjacent organs, it is movable. When adherent to the liver or diaphragm, it moves freely with respiration. Such a tumor, occurring in persons past the age of forty years, is almost certainly cancerous. In younger persons it may be suggestive of hypertrophy of the pylorus. Important as age is in deciding the nature of the case, too much dependence must not be placed upon it.

Tumor of the anterior wall of the stomach rarely escapes detection, even in its earliest stages. It changes its position according to the degree of distention of the stomach. One should not, however, ignore the pylorus as the seat of lesion simply because the tumor does not happen to be in the ordinarily-accepted position for pyloric cancer. Very exceptionally it may be found to the right of the median line below the level of the umbilicus. When the stomach is displaced, it is possible for pyloric tumor to present itself as a prominence to the left of the median line. Occasionally mistakes may be made respecting the character of the growth because the pulsations of the abdominal aorta are felt through it.

The fact that the tumor changes its position after distention of the stomach is of itself of diagnostic value. For example, a tumor that is readily palpable when the organ is empty, but disappears after distention, is almost certainly situated in the posterior gastric wall.

3. **Percussion.**—A proper understanding of the value of percussion as a means of diagnosing gastric disorders demands a consideration of facts bearing on the varying physical conditions of the normal stomach. It must be remembered that the stomach, aside from its cardiac extremity, is not a fixed organ, altering its position by reason of its different degrees of distention, whether by food or gases. Then, too, it is readily displaced by pressure, as from pleuritic effusion, ascites, and abdominal tumors. It should also be kept in mind that there is no standard by which the actual normal capacity of the stomach can be gauged; 2000 cubic centimeters have been assigned arbitrarily as the limit beyond which it cannot go without being designated large or dilated. At the same time, such a large capacity is abnormal in many individuals. In each case one should endeavor to determine what is the normal of the individual. Another difficulty arises from the variations in the size of the stomach in cases in which its walls are relaxed.

It is now in order to detail the normal outlines of the stomach as obtained by percussion. As this organ always contains gas, percussion elicits a clear, low-pitched, tympanitic note. This will be modified by the presence of solids or liquids, or by undue flatulent distention; in the latter case, the sound assuming a metallic quality.

Inasmuch as a large portion of the stomach is situated beneath the liver and diaphragm, its upper limits cannot be discovered by percussion. Normally there is an area which lies within our capabilities, and this area may be described as follows: (*a*) *Traube's semilunar space*. This is an area of tympanitic resonance included between the following lines: Above, a slightly arched line from the sixth intercostal cartilage in the parasternal line to the ninth in the mid-axillary line; to the left by the anterior edge of the spleen; and below by the left costal margin. Briefly, it covers that portion of the stomach which lies in contact with the thoracic walls. Above, this area is to be delimited from pulmonary structure, which is best done by percussing from stomach to lung. (*b*) Anteriorly is a portion of the abdominal walls, beneath which lies the anterior wall of the stomach, and this area gives gastric tympanitic resonance. On the left it is bounded by the semilunar space just described. On the right it is limited by the hepatic dullness; while below, and following a curved line, it extends to one or two finger-breadths above the umbilicus.

The organs, then, which are adjacent to the stomach, and the points of contact which are to be delimited by percussion, are the lungs, the liver, the spleen and the colon. The percussion-outlines separating the stomach tympany, on the one hand, from the characteristic sounds of the lungs, liver and spleen on the other, can vary only as these organs assume a pathological state. The lower limit, that separating the stomach from the colon, is the most important, and at the same time the most difficult to determine. Still, the problem is one capable of satisfactory solution if care is practised. Ordinarily the percussion-note over the colon is higher in pitch than that over the stomach. Sometimes, however, the stomach contains but little gas, and the colon is greatly distended, and then one will encounter some difficulty in reaching a decision. Even then a practised ear can detect a line at which the percussion-note changes, and which in all probability marks the line of the greater curvature of the stomach. If the physician wishes to be positive as to the result, he may distend the stomach with carbonic acid gas, when all difficulty will disappear; or he may have the patient drink a full glass of water, and, percussing with the patient in the upright posture, will discover a line of dullness marking the lower boundary of the stomach. If this line is lower than normal, he may make further application of this procedure to determine whether the stomach is dilated or displaced downwards. In the case of displacement or relaxation of the gastric supports, the line of dullness will be found to be still lower, on percussion, after the patient has taken a second glass of water.

Auscultatory percussion is ofttimes more accurate than simple percussion in delimiting the outlines of the stomach. The chest-piece of the stethoscope should be placed over the fundus. Then percussion should be practised, starting at a point some distance from the instrument and gradually approaching it until the sound of gastric tympany is obtained. Then the same procedure is repeated again and again, each time in a different line, centering in the position of the bell of the stethoscope. Auscultatory percussion is especially valuable in determining whether a tumor discovered by percussion or palpation belongs to the stomach, as the dullness produced over it in that case presents important differences from what is observed in cases of hepatic and gall-bladder enlargements.

Diminution of the area of gastric tympany results from encroachment of neighboring organs and tissues. The following clinical conditions are to be mentioned in this connection :

(i) Enlargements of the spleen.
(ii) Enlargements of the left lobe of the liver.
(iii) Cardiac hypertrophy and dilatation.
(iv) Pericardial effusion.
(v) Left-sided pleural effusion.

Diminished gastric tympany below can occur only when the stomach is below the normal standard in size, or, exceptionally, when the organ is maintained in an abnormal position by adhesions. Diminution in the size of the stomach can be diagnosed with certainty after the organ has been distended with carbonic acid gas.

In **left-sided pleural effusion,** one of the earliest physical signs is dullness in Traube's semilunar space. As the effusion increases, the dullness extends, until it finally may involve the entire area. In croupous pneumonia there is no alteration in the percussion-sounds over Traube's space.

The **gastric tympanitic area is increased** above by causes outside of the stomach, and these relate entirely to disease of adjacent organs. They are as follows :

(i) Cirrhosis and other diseases of the liver, attended by diminished size of that organ.

(ii) Retraction of the lung, dependent upon old pleurisies and other inflammatory diseases.

(iii) Flatulent distention and dilatation, which may bring about displacement of the thoracic viscera ; the increased gastric tympany in the upward direction being manifest especially in Traube's semilunar space.

In dilatation and prolapse of the stomach, the increased tympany is found in a downward direction, in addition to the increase in Traube's space.

4. **Auscultation.**—While the value of auscultation in the diagnosis of diseases of the stomach is not as great as the procedures already mentioned, nevertheless it gives, at times, information that is by no means unimportant ; hence it should never be neglected. Systematic auscultation should take cognizance of the following :

(*a*) **The Deglutition Sounds.**—These are two in number, known as the first and second deglutition sounds. They are best heard with the stethoscope over the ensiform cartilage. The first sound takes place at the moment of swallowing. It is very often absent. The second sound follows the first after an interval of seven or eight seconds. When present in their normal relations, we are justified in assuming that the cardiac orifice is normally patent. When both sounds are absent, we may suspect, other phenomena confirming the suspicion, that there is stenosis at the cardiac orifice.

During the act of drinking, auscultation discovers trickling sounds, caused by the water rolling down the sides of the stomach. By carefully locating these sounds, one is enabled to judge of the size and location of the stomach. This procedure can hardly be said to have very great value, as other measures of easier application give us the necessary information. The same may be said of Rosenbach's suggestion, namely, that the stomach be partly filled with water, and a tube introduced. Air is then blown into the stomach, with the end of the tube in the water. Auscultation discovers the bubbling, and by changing the depth of the tube, one judges of the size of the stomach. There can hardly be any doubt of the reliability of the information imparted by this procedure; but, as already stated, it is not a method of practical value in the consulting-room.

(*b*) **Friction Murmurs.**—When the peritoneal surfaces have been roughened by inflammation, friction murmurs may be heard during the movements of respiration.

The respiratory movements may produce another sound when the stomach is greatly distended with gas. The parietal peritoneal surface, gliding over the distended visceral membrane, sets up vibrations in the latter, which have been compared to the sounds of a violoncello.

(*c*) **Sizzling Sounds.**—The name describes the character of the sounds. Their presence indicates that food is stagnant and undergoing fermentation in the stomach.

(*d*) **Succussion and Splashing Sounds.**—(i) A splashing sound which is produced by the movements of the stomach as it rises and falls with respiration. (ii) A sound, first described by Hippocrates, elicited by shaking the patient, and dependent for its existence upon the presence of air and liquid in the stomach, if that organ is enlarged. It does not afford much information. The same sound may occur when the patient turns over in bed suddenly.

(*e*) **Gurgling Sounds.**—These may be heard a distance from the patient. In one group of cases the sounds are produced by contraction of the stomach when that organ contains nothing but air or gas, as when one is hungry. It is sometimes called the growling stomach.

(*f*) **Ringing sounds,** due to the action of the stomach as a resonating chamber in transmitting the sounds of the heart, possess no clinical significance.

The lowermost limit at which splashing sounds can be heard after drinking should never be below the umbilicus. This splashing sound is an evidence of atony. The greater the atony, the smaller the quantity of liquid necessary to produce the sound.

5. **Examination of the Gastric Contents.**—Valuable as is the information to be obtained by chemical examination of the gastric contents, this procedure should not be resorted to indiscriminately. It is indicated

only in cases in which evidence as to certain secretory conditions is wanted. It is foolish to resort to it for the purpose of adding to the diagnostic procedures already practised on the patient. Accidents have been known to follow the use of the tube ; hence the condition of the patient should be thoroughly understood before starting the examination. The conditions which contra-indicate its use are the following :

(*a*) **Heart Diseases, with Failing Compensation.**—The violent gagging and vomiting which may be excited is capable of adding to the difficulties of an already overburdened heart, producing cyanosis, and even death.

(*b*) **Aortic aneurysm** is a contra-indication, for the same reason that it is a contra-indication to the passage of the sound in œsophageal obstruction. It may produce rupture.

(*c*) **Advanced Arterial Degenerations.**—It is not advisable to use this method of examination in persons of advanced years with atheromatous vessels, because of the liability to rupture during violent attacks of vomiting. For the same reason, when a patient has recently had an apoplectic seizure the measure is contra-indicated, as the bleeding may be renewed. Fortunately the examination is rarely called for in such cases.

(*d*) **Nor should it be employed in the advanced stages of any chronic disease, as tuberculosis, etc.** Again, it is difficult to see why this warning is necessary ; but it is a fact that some physicians have a rage for carrying examinations to an extreme, and have been known to use the tube as a routine measure.

(*e*) The tube should never be used during the course of **febrile affections.**

(*f*) It should never be employed in cases of **suspected ulcer, unless it is important to make a differentiation from cancer.** Under no circumstances should the tube be introduced when there has been recent hæmatemesis.

(*g*) In **acute gastritis** the diagnosis is self-evident, without the examination. The stomach irritability, too, will be increased by the tube. The local condition calls for complete rest of the parts.

A proper examination of the gastric contents demands that the materials examined be obtained under fixed and unvarying conditions. Hence the gastric contents should be obtained under fixed and varying conditions, which can only be when removed at a definite hour after taking a standard meal. It will not do to wait for the patient to vomit in order to obtain a specimen, although conditions are conceivable under which we are content with this makeshift. Vomited specimens may be obtained at a time when the contents are not in fit condition for examination, so that the information obtained is unreliable. Various test-meals have been proposed. The one most commonly used is the simplest, and is known as

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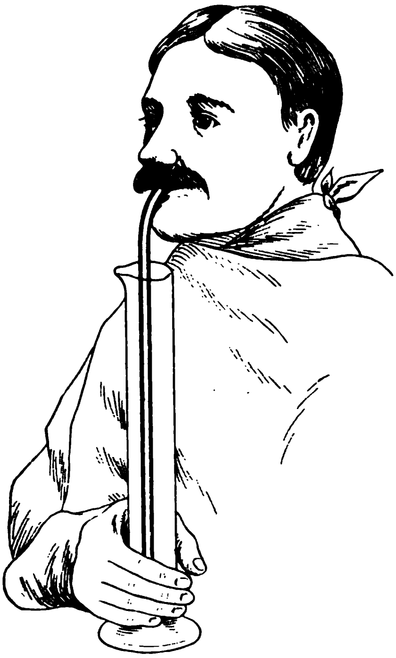
Ewald's Test-Breakfast.—The patient takes in the morning, when the stomach is empty, a breakfast consisting of a roll and a half-pint of plain tea or water. The gastric contents are ready for examination one hour after the meal.

Leube and Reigel's test-dinner is also popular, though much less frequently used than the above. The patient takes about one-half pint of soup, a piece of beef or beefsteak, some potatoes, and a roll. The examination is made three to four hours later.

Hemmeter advises a double test-meal, as follows: 8 A.M., 1 small piece of beef, scraped and broiled, 80 grm.; 30 grm. boiled rice; 1 glass of milk and a piece of bread. Four hours later an Ewald's test-breakfast is given. The advantages claimed for this method is that "we may, in a large number of instances, recognize conditions of gastric motility and secretion before we analyze the contents. For instance, total disappearance of the entire breakfast meal points to a normal digestion. Absence of all proteids—beef and egg—and presence of considerable carbonates—rice and bread—points to hyperchlorhydria; and, again, absence of carbohydrates and presence of some of the beef and egg points to hypochlorhydria, subacidity, or anacidity. Presence of the entire meal, with perhaps milk uncurdled, means impaired motility with atrophy of gastric mucosa, absence of acid, enzymes, and pro-enzymes. If the entire breakfast has disappeared, the status of the gastric secretions may be ascertained from the Ewald test-breakfast, which is still present." Notwithstanding the greater inconvenience of the preparation for this meal, it will be seen that there are numerous cases in which its use will be demanded.

FIG. 40.



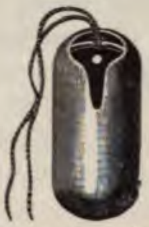
Showing position of receptacle when removing gastric contents by expression. The necessary retching accompanying the act causes the patient to assume other attitudes than the easy one seen in the cut.

To **withdraw the gastric contents** for examination we may resort to one of the following methods:

a. Expression or aspiration through the stomach-tube.
b. Einhorn's stomach-bucket.
c. Spallanzani and Edinger's sponge method.

(*a*) *Expression or aspiration through the stomach-tube.* The tube used for this purpose should be a short stomach-tube having large side- and end-openings. To its outer end is attached a piece of glass tubing about four inches in length. The instrument is passed in the manner explained when describing the use of the œsophageal sound. Then the outer end, with the glass tube attached, is placed in a bottle which is held by the patient, who is now instructed to bear down as if at stool. At the same time the physician gives him some help by exerting firm pressure on the epigastrium. These combined manipulations are followed by prompt expulsion of the gastric contents. Sometimes the presence of the tube excites retching sufficiently severe to bring up the necessary quantity through the tube without any effort on the part of the patient or physician.

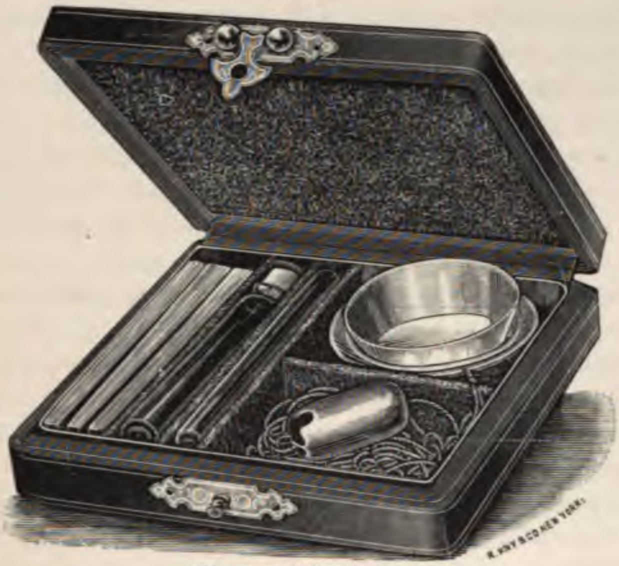
FIG. 41.



Einhorn's stomach-bucket.

Sometimes the expression method fails, though it has never been my experience to have it do this. Then we must resort to aspiration for removal of the stomach contents. For this purpose we may use a Politzer bag attached to the outer glass tubing, or a specially-constructed bulb-apparatus. I must say, however, that with a very little experience, one can nearly always obtain the gastric contents by simple expression. Aspiration brings with it increased danger of damaging the gastric mucous membrane.

FIG. 42.



The stomach-bucket set.

(*b*) *Einhorn's stomach-bucket* is a small silver vessel similar in shape to the gelatine capsules in common use. It is one and three-quarter inches long by three-quarters of a cm. wide. It has large perforations at the top, to one of the partitions of which is attached a silken thread. Before introduction, the instrument is thoroughly moistened by dipping it into waters so that it will take up the gastric contents more readily. The bucket is next placed in the pharynx, and the patient is directed to swallow a few times. It usually requires one or two minutes for it to reach the stomach, which may be known when the silken string has been played out a distance of sixteen inches. The instrument is permitted to remain in the stomach for five minutes, by which time it is usually filled with the gastric contents. Then it is withdrawn. Just as it reaches the cardiac orifice a sense of resistance is felt. This may be overcome by directing the patient to swallow. The advantages claimed for this method of obtaining the specimen are that it does not cause the patient much inconvenience, is devoid of danger in cases in which ulcer is suspected or where there has been recent hæmatemesis, and that it is especially useful for the general practitioner who desires the specimen for making a simple test for the presence of hydrochloric acid. Its objections are the small quantity of contents obtained, the liability of the tube being filled with œsophageal mucus during its introduction, and the inability of some people to swallow it. The clogging of the instrument with mucus may be obviated by covering the openings with gelatine, which dissolves, after a short time, in the stomach.

(*c*) The *sponge method* is open to all the disadvantages of the stomach-bucket, besides introducing additional ones of its own. The sponge is squeezed out during its withdrawal, and hence is deprived of much of its contents. Then, too, it also absorbs the contents of the pharynx and œsophagus. The specimen obtained cannot, therefore, be even moderately pure.

The gastric contents having been obtained, they are next examined as to the following points:

i. Quantity.
ii. Chemical reaction.
iii. Total acidity.
iv. Presence of free hydrochloric acid.
v. Presence of free lactic acid.
vi. Propeptone.
vii. Pepsin.
viii. Peptone.
ix. Rennet ferment.
x. Dextrin.
xi. Erythrodextrin.
xii. Achroödextrin.
xiii. Maltose.

(i) **The Quantity.**—Providing the stomach is empty at the time of taking the test-breakfast, as it always should be in the morning, the quantity of gastric contents obtained through the tube should be not over 40 c.c. nor under 20 c.c. If the quantity greatly exceeds these figures, we may infer the presence of hypersecretion; or, if no contents are obtainable, that there is undue patency of the pylorus.

(ii) **The Chemical Reaction.**—The reaction of the gastric contents is normally acid; rarely is it found otherwise, even in disease. The acidity is determined by means of litmus paper, which is turned red in the presence of an acid reaction.

Litmus, however, is of use only to determine an absolute acidity. It reacts whether the acidity is dependent upon free acid or acid salts. For the determination of free acids the following reagents are recommended:

A weak solution of congo-red is turned blue by hydrochloric acid, and violet by organic acids.

Benzo-purpurin solution turns dark-blue in the presence of hydrochloric acid, and brownish-black in the presence of butyric or lactic acid.

Clinically, this test possesses but little importance. It is otherwise, however, with the degree of acidity,—*total acidity*, as it is called.

(iii) To **determine the total acidity**, the following reagents and apparatus are necessary: A decinormal solution of sodium hydrate, a one per cent. alcoholic solution of phenol-phthalein, a graduated burette, burette holder, and beaker. Then 10 c.c. of the filtered gastric contents, to which has been added one drop of the phenol-phthalein solution, are placed in the beaker. The burette is filled with the decinormal sodium hydrate solution, which is permitted to mix with the gastric contents drop by drop. As each drop falls into the beaker it produces a red color, which is speedily dissipated at first. Finally there comes a time in the course of the process when the red reaction remains permanent. Then the process is stopped, and the quantity of sodium hydrate required is read off on the burette. This figure is multiplied by 10, and the result gives us the total acidity. To illustrate: If 7.5 c.c. of the sodium hydrate solution are required to produce the final reaction, then the total acidity is 75. The percentage amount of hydrochloric acid may be calculated from this figure by multiplying it by 0.00365. Thus, the acidity being 75, the percentage of hydrochloric acid will be 75 x 0.00365, or .27275 per cent.

Significance of Alterations in the Total Acidity.—*Diminished acidity*, as a rule, indicates diminished gastric secretion, especially of hydrochloric acid. It may occur in organic diseases of the stomach, or in constitutional diseases in which the gastric secretion is in abeyance. Hence it may be noted in fevers; various blood diseases, as chlorosis, pernicious anæmia, etc., and chronic wasting disorders; and, among the organic diseases of the stomach, those associated with atrophy of the gastric mucous membrane, as chronic gastric catarrh and cancer. It has been claimed that diminished acidity may be symptomatic of a neurosis. Such cases are open to doubt, and the diagnosis should be reserved.

Increased acidity may be dependent upon increase of the hydrochloric acid or of the organic acids. The significance of hyperacidity must depend, therefore, upon the particular chemical element to which it is due. The reader is therefore referred to later paragraphs in which the special indications attendant upon increase of hydrochloric acid or the presence of lactic, butyric and acetic acids are described.

Normally, from four to six cubic centimetres of decinormal solution of sodium hydrate are required for the reaction, making the normal total acidity vary from 40 to 60.

If the gastric contents are alkaline, the degree of alkalinity may be determined by mixing the gastric contents with the phenol-phthalein solution as before; but now the burette should be charged with a decinormal solution of hydrochloric acid. The analysis proceeds as before. When sufficient of the acid is added to the gastric contents to dissipate the red reaction of the phenol-phthalein with the alkaline fluid, the process is terminated, the quantity of hydrochloric acid used is noted, and the result multiplied by 10. The result indicates the alkalinity.

The significance of alterations in the total acidity varies according to the variations in the different elements which go to make it up.

(iv) **The Presence and Quantitative Determination of Free Hydrochloric Acid.**—Four tests are in common use for determining the presence of free hydrochloric acid in the gastric contents. Günzburg's is the favorite with the majority of clinicians. The reagent is prepared as follows:

Vanillin, ...	1 part.
Phloroglucin, ...	2 parts.
Alcohol, ...	30 parts.

It is claimed by some that the test is made more delicate by using a more dilute solution, *i.e.*, by taking 100 instead of 30 parts of alcohol. Günzburg's solution is perishable. Still, it may be kept for a year or more if placed in dark-glass bottles, protected from light and air. To make the test, place one or two drops of the gastric contents on a porcelain capsule, and add one or two drops of the reagent. Then heat gently over an alcohol flame. If free hydrochloric acid is present, there will appear bright-red or carmine lines or patches, especially marked at the periphery of the dried materials. The quantity of free hydrochloric acid may be determined roughly by the intensity of the reaction. It is claimed that Günzburg's reagent will detect 1 part of hydrochloric acid in 20,000 parts of diluent.

Dimethylamidoazobenzol is as delicate as the Günzburg test. A one-half of 1 per cent. solution in alcohol, known as Töpfer's solution, is employed. One drop of this, added to some of the filtered gastric contents, produces a rose-red color if free hydrochloric acid is present.

Boas's resorcin test is a favorite with many. The reagent is prepared as follows:

Resorcin,	5.0
Sacchar. albi,	3.0
Alcohol, ad,	100.0

The test is made in the same way as that of Günzburg. The result is a cherry-red reaction. The test is a reliable one, but less sensitive than that of Günzburg. It has the great merit of cheapness. The solution, moreover, keeps well.

Tropæolin 00 is also recommended by Boas. A few drops each of a saturated solution of tropæolin 00 and gastric contents are placed in a porcelain capsule and well smeared over the sides of the dish by rotating it. The whole is heated over an alcohol flame. If free hydrochloric acid is present, there will appear lilac-blue to blue streaks.

The actual quantity of hydrochloric acid present may be determined from the total acidity as described in a previous paragraph; or, one of the following methods may be employed.

Günzburg's reagent, as already stated, is capable of determining 1 part of hydrochloric acid in 20,000 parts of diluent. This fact may be utilized by diluting the gastric fluid until the reaction can no longer be obtained. If, for example, there is no reaction after diluting beyond five times, hydrochloric acid is present in the proportion of 5 to 20,000, etc. This method is rather tiresome, hence is seldom employed.

Töpfer's dimethylamidoazobenzol method is both reliable and easy of application. Two or three drops of Töpfer's solution are added to 10 c.c. of gastric contents. A decinormal solution of sodium hydrate is placed in a burette and permitted to mix with the gastric contents drop by drop. As soon as a sufficient quantity of the alkali to neutralize the free hydrochloric acid is used, the rose-red reaction of the Töpfer solution with the gastric contents disappears. The quantity of sodium hydrate used is then noted, and multiplied by 0.00365. This gives the percentage of free hydrochloric acid.

The determination of the actual quantity of free hydrochloric acid does not always lead to accurate conclusions, because a certain quantity of that reagent is apt to combine with the albuminous and other substances found in the stomach. Hence there are times when a knowledge of the amount of combined hydrochloric acid is important. This may be obtained by an extension of the Töpfer method, just described.

A few drops of a 1 per cent. watery solution of alizarin mono-sulphonate of sodium are added to 10 c.c. of gastric contents. This mixture is titrated with a decinormal solution of sodium hydrate until a clear violet reaction is obtained. This result occurs only when sufficient alkali has been added to neutralize the acidity of hydrochloric acid, organic acids, and acid salts. Combined hydrochloric acid has no effect on it. Now, if we subtract the acidity as obtained when determining the amount of free hydrochloric acid from the result just obtained, we determine the amount of acidity dependent upon organic acids and acid salts.

Significance of Alterations in the Secretion of Hydrochloric Acid.—Diminution in the amount of free hydrochloric acid means alterations in the secretory functions of the gastric mucous membrane, and generally signifies atrophy of the mucous membrane. In the early days of the clinical study of the chemistry of the stomach, absence of free hydrochloric acid was heralded as an unfailing sign of cancer of the stomach. It is now recognized that hydrochloric acid is absent in cases of gastric cancer only when the disease has caused atrophy of the glandular structures. This, it is true, is a common result, but by no means an invariable one. The gastric disorders attended by *diminished or absent secretion of free hydrochloric acid* are as follows:

Cancer.
Chronic gastric catarrh.
Atrophy of the stomach.
Achylia gastrica. (Absence of gastric secretion.)

The statement that hydrochloric acid may be entirely absent from the gastric contents in some cases of hysteria and neurasthenia is, in my opinion, sadly in need of confirmation. In all such seen by me, notwithstanding the neurotic character of the associated symptoms, the subsequent histories of the cases proved the anachlorhydria to be due to organic changes.

Increased production of hydrochloric acid (hyperchlorhydria) occurs in

Irritative dyspepsia.
Gastric ulcers.
Neuroses of the stomach.

v. **The Presence or Absence of Lactic Acid.**—The best test for the presence of lactic acid in the gastric contents is Uffelmann's, the reagent for which is prepared as follows: Add a few drops of pure carbolic acid to a weak solution of ferric chloride, the result being an amethyst-blue fluid. To the reagent as thus prepared a few drops of gastric contents are added, and if lactic acid be present, the solution will change to a canary yellow. The fatty acids give an ashy-gray reaction; inorganic acids decolorize the fluid.

The reaction of lactic acid with Uffelmann's reagent may be simulated by the presence of phosphates. To avoid this difficulty, the lactic acid may be separated from the gastric contents by shaking the latter with double the quantity of ether. The mixture is then permitted to settle, when the ethereal solution is removed and the ether evaporated. A watery solution is made of the residue, and the Uffelmann test is applied as before.

Arnold suggests a test which he declares to be reliable, and free from error. Two solutions are employed: (*a*) A 0.2 c.c. of a saturated solution of gentian violet in 500 c.c. of distilled water; and (*b*) tincture of the chloride of iron, 5 c.c. in 200 c.c. of distilled water. To 1 c.c. of the gentian violet solution add 1 drop of the ferric chloride, the result being a blue fluid. To this mixture add a few drops of gastric contents. If lactic acid is present, a yellowish or greenish color will appear.

Kelling's Test.—To 2 c.c. of gastric contents are added ten times that quantity of water. To this mixture are added one or two drops of a 5 per cent. aqueous solution of perchloride of iron. If lactic acid is present, a distinct green color will be perceived on holding the tube against a white background.

A quantitative estimate of the lactic acid in the gastric juice need not be exact for clinical purposes. Valuable approximate results are obtainable by Strauss's method. For this purpose a mixing funnel has been devised. At its lower end is a small stopcock. The body of the funnel is marked at 5 c.c., and again at 25 c.c. The funnel is filled to the 5 c.c. mark with the filtered gastric fluid, and then ether is added to the 25 c.c. mark. The whole is well shaken, and then allowed to separate, when the fluids are permitted to run out until the 5 c.c. mark is reached. Next distilled water is added, to 25 c.c., and two drops of tincture of chloride of iron added, 1 : 10. The mixture is well shaken. If lactic acid is present in greater proportion than 1 in 1000 a marked green color will be produced; if in smaller proportion, a pale-green hue.

Significance of the Presence of Lactic Acid.—When lactic acid is found in the gastric contents, it must have been introduced in the food or it must have been formed by fermentation-changes. In the latter case it constitutes an important sign of cancer, being present in the majority of cases of that disease. It is absent, however, when the power of the stomach to empty itself is unimpaired. If, however, lactic acid is found to be present and hydrochloric acid absent, and especially if there be impairment of the motor-power of the stomach, it is fairly certain that cancer of the stomach exists, although no tumor is palpable. The absence of lactic acid must not be accepted as disproving the presence of cancer.

Lactic acid is found normally only when introduced in the food, as after a meal of meat. Small traces are obtainable after Ewald's test-breakfast. If absolute accuracy is demanded, in testing for the acid it is necessary to give a test-meal which will not yield the lactic acid. For this purpose Boas has recommended the following: One or two litres of oatmeal gruel, to which a little salt has been added. The gastric contents are to be obtained by expression one hour after taking the meal.

vi. **The Detection of Propeptone.**—The most readily-applied test for the presence of propeptone is the addition of some saturated solution of common salt to an equal quantity of gastric contents. If propeptone is present, a turbidity proportionate to its amount will appear. Sometimes the reaction will not take place. Then one or two drops of acetic acid are to be added to the mixture; in which case, if propeptone is present, the turbidity will appear. This cloudiness is dissipated by heat, but the precipitates form anew on cooling.

vii. **The Detection of Peptone.**—The mixture resulting from the preceding test for propeptone is filtered. The filtrate is made strongly alkaline by adding sodium hydrate solution. Next a few drops of a 1 per cent. solution of cupric sulphate are added. If peptone is present, there will occur a purplish or violet-red reaction.

viii. For the **detection of pepsin,** take a disc of coagulated white of egg, 1 c.c. in diameter and 1 mm. in thickness. This is placed in 5 c.c. of filtered gastric contents in a test-tube, and kept at the temperature of the body for a number of hours. If pepsin is present in normal quantity, the disc will have been dissolved at the end of two to six hours.

ix. **Rennet Ferment.**—To determine the presence of the rennet ferment, three or four drops of the gastric filtrate are added to about 5 c.c. of milk in a test-tube. The mixture is then kept at blood-heat in a cup of warm water (temp. 100° F.) for about fifteen minutes, when, if the ferment is present in normal quantity, the milk should be curdled. In order, however, to be positive that it is present, the process should be continued for an hour without producing coagulation of the milk.

To determine the presence of **rennet zymogen,** the mixture used in the experiment just described has added to it a few drops of a 1 per cent. calcium chloride solution, and is permitted to stand for a few minutes. If rennet zymogen is present, coagulation will take place in a few minutes.

x. **Examinations for peptone, propeptone, pepsin,** etc., simply reveal the fact that the gastric juice is of normal quality and quantity. The information obtained does not afford information of sufficient clinical value to demand their routine use in the examination of the gastric contents.

xi.–xiii. **Erythrodextrin, Dextrin, Achroödextrin, and Maltose.**—The same reagent is used for determining the presence of each of these substances, which are all products of starch digestion. This reagent is made as follows:

Iodine, ...	0.1
Potassium iodide, ...	0.2
Aquæ destillata, ...	200.00

A few drops of this are added to the filtered gastric contents. If dextrine be present, the fluid is turned blue; if erythrodextrin, a red color is produced; achroödextrin changes it to a yellowish hue; while maltose effects no change in the color of the reagent.

These reactions are mainly of importance in determining the extent of starch digestion in cases of hyperacidity or increased gastric secretion. The starches should be digested completely one hour after taking into the stomach; hence, normally, the iodine should not be changed in color after this time.

To Determine the Absorptive Power of the Stomach.—The favorite method for determining the absorptive power of the stomach is that suggested by Penzoldt and Faber. The patient takes three grains of iodide of potassium in a gelatine capsule. Care is taken that none of the drug is on the outside of the capsule, otherwise it will contaminate the saliva. After a short time, the saliva is tested for iodine every minute or so in the following way: Strips of paper saturated with starch and dried are moistened with the saliva, and then touched with fuming nitric acid. If iodine is present in the saliva, a blue reaction takes place. Normally, this should appear in six and a half to ten minutes.

Herschell's method requires that the patient take three grains of powdered rhubarb in a capsule. If the absorptive function of the stomach is normal, the rhubarb will be manifest in the urine in fifteen minutes, it being detected in that fluid by a red reaction with liquor potassæ.

To Determine the Motor-Power of the Stomach.—This question resolves itself into the determination of the ability of the stomach to empty itself within a reasonable time after a meal. For all practical purposes we may rest satisfied with the motor-function of the stomach if the organ is empty in the morning on waking. Leube's test takes cognizance of this fact. He suggests that the stomach be washed out about six or seven hours after a meal. If at that time no food-particles are found in the washings, the motor-power may be regarded as normal.

Ewald's salol-test depends upon the fact that this drug when exposed to the action of alkaline fluids is decomposed into salycylic acid and phenol. The acid gastric contents have no effect on the salol as long as it remains in the stomach. As soon as it enters the intestinal tract, decomposition takes place. The salicylic acid is absorbed, and is excreted by the kidneys as salicyluric acid. Its presence in the urine may then be detected very readily by the addition of a few drops of a ferric chloride solution, which strikes a violet color. Before making the test, the patient should be instructed to empty his bladder. He then urinates every half-hour for about two hours. The different specimens thus obtained are tested with the ferric chloride. Under normal conditions, the reaction should take place in all the urines passed after one hour.

Huber suggested that the time required for the disappearance of the salicyluric acid from the urine is a more valuable guide of the gastric motor-power. Normally, the excretion of salicyluric acid by the urine should continue for 24 hours. When the motor-function is weakened it may be prolonged for 48 hours, or even longer.

Klemperer's oil-test is too unpleasant and too troublesome for clinical use. It need be mentioned only to be discountenanced.

There may be distinguished from the motor-function of the stomach, by which is meant the ability of the organ to empty itself within the normal period after taking food, the mechanical function, by which is understood the various movements which bring about an intimate admixture of the food with the digestive fluid, *i.e.*, the gastric juice. Two elements enter into the mechanical function, namely, the peristaltic movements of the stomach, and the passive movements effected by inspiration and expiration. This function cannot be investigated excepting by the aid of specially-devised apparatus, of which two have been proposed, namely, Einhorn's gastrograph and Hemmeter's deglutible elastic bag.

Einhorn's Gastrograph.—This instrument may be described best in its inventor's words: "The whole apparatus comprises: 1. The ball (being the principal part). 2. A few electric calls. 3. The ticker.

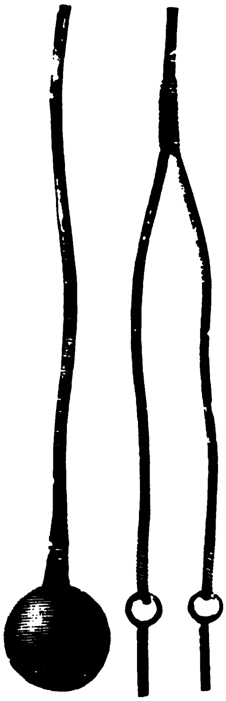
"The ball (Fig. 43) consists of two hollow metallic hemispheres (*a*), which are screwed together; within it is lodged and attached to the upper hemisphere, but perfectly insulated from the same at the attachment, another ball, provided with spikes (*b*), radiating in all directions, but not touching the inside walls of the hemispheres; another very small platinum ball (*c*) lies within the large ball and can be freely moved in all directions, knocking at the spikes (see Fig. 44). Two insulated wires—one connected with the hollow ball, the other with the spiked ball—are encased in a very fine, thin rubber tube, forming the cable, and separated at the end into two branches, which must be attached to an electric battery. As soon as the platinum ball touches one of the spikes an electric circuit is made; when, however, the platinum ball moves a little way and ceases to touch the spike, the current is broken. At each motion of the ball apparatus a rolling of the platinum ball takes place, and the electric current is either closed or broken. When the apparatus is at rest there is no change in the current. On connecting the 'ticker' with the battery and the ball, each motion of the latter will be recorded on the paper, showing the 'breaks' and 'makes' of the current.

"If the ball is swallowed and brought into the stomach, the motions of the former—which are caused by the active and passive movements of the stomach—can be recorded in the way described.

"The strip of paper which has rolled off from the roll is cut off, and the marks are then perused. The black line shows when the current was closed, the empty places where there was no current. As an instance, I (Einhorn) give a few gastrograms reduced ten times. It is practical to enter the marks of the strips into a copybook. This is done in the following way: Each line is divided into three equal spaces, each space corresponding to one minute, and the 'breaks' and 'makes' of the current marked with dots at the corresponding place. In this way the number of current-changes can very easily be looked over and comparisons made.

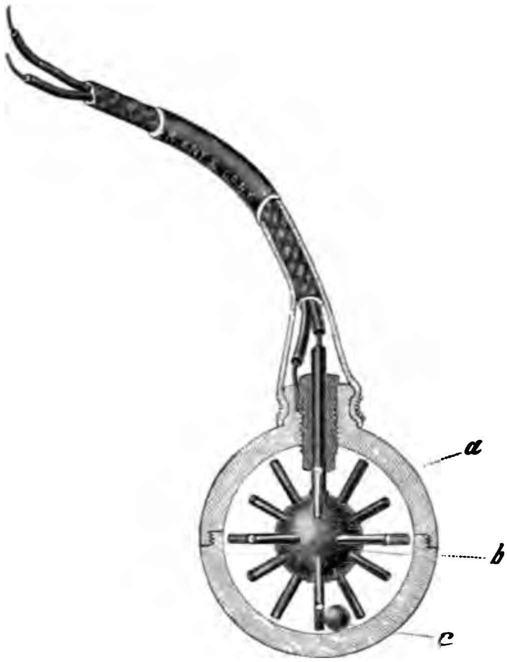
"(*a*) I (Einhorn) have made several tests with the gastrograph on healthy people. The experiments show that the stomach is not so inactive, mechanically, as several authors believed, and that it churns the contents almost continuously, with slight periodical interruptions. The number of motions for three minutes averaged from three to forty-one. When fasting, the mechanical action of the stomach seems to be much less than after meals.

FIG. 43.



The ball apparatus of Einhorn's gastrograph, actual size.

FIG. 44.



Cross-section of the ball, showing its interior construction. Enlarged three and a half times. *a*, the two hemispheres; *b*, the spiked ball; *c*, the platinum ball.

"(*b*) Pathological. Most patients have been examined with the gastrograph either when fasting or from an hour to an hour and a half after the test-breakfast, taking about half a glass of water when swallowing the ball; many of the patients have been examined under both conditions on different days. Some of them have been subjected to a very great number of tests, in order to ascertain whether there is a certain constancy as to the results. The whole number of patients examined was twenty-seven, the number of tests sixty-four.

" In perusing the gastrograms, there are three different classes among them. One corresponds to the normal; the second class is marked with too much mechanical action, the number of dots being greatly increased; the third class shows a remarkable slowness and sluggishness of the mechanical functions, the number of dots being reduced to 4, 3, or 0."

Hemmeter's Method.—This is a modification of the method proposed by Moritz. The latter used a spherical rubber bag, which was introduced into the stomach and then inflated. By means of a manometer or kypograph, alterations in the intragastric pressure due to peristaltic movements were chronicled. Hemmeter's modification consists in using a stomach-shaped bag. There can be no doubt that this procedure is a most excellent one for the purpose for which it is designed. At the same time, it is one which seems better adapted to the laboratory than to the consulting-room, notwithstanding the enthusiastic advocacy of its inventors.

The Significance of Altered Mobility of the Stomach.—The motor-power of the stomach may be increased or decreased.

It is increased only in cases of certain motor neuroses.

Misdirected gastric motor activity is found in other neuroses of the stomach, including

Cardiospasmus.
Regurgitation.
Rumination.
Nervous vomiting.
Eructations.

Diminished motor-power may occur in connection with

Gastric atony.
Gastrectasia.
Pyloric obstruction.

The Microscopic Examination of the Gastric Contents.—The information obtainable by microscopic examination of the gastric contents is limited to the discovery of certain organisms, as the sarcinæ ventriculi and the Oppler-Boas bacillus, and the recognition of detached portions of tumor and gastric mucous membrane.

The *sarcinæ ventriculi* appear as squares or tetrahedra divided by lines so as to resemble bales of cotton, in groups or colonies. In extreme cases, the gastric contents may consist of an almost pure culture of this organism. They may be observed in small numbers in health; but when present in large numbers they are significant of fermentative processes, as in gastric dilatation. It is worthy of note that although, in cancer of the stomach, the physical conditions are apparently such as to favor their development, they are rarely present in the advanced stages.

The *Oppler-Boas bacillus* is almost constantly present in carcinoma of the stomach, and in association with the presence of free lactic acid. It is a non-motile micro-organism, of unusual length, readily discernible by objectives of low power. Sometimes the individual organisms are joined together end to end, forming long threads.

In obscure cases of gastric disorder the stomach-washings should be carefully searched for solid particles, which may be portions of the gastric mucous membrane or of tumors. In the former case, one is led to believe in the presence of superficial gastric erosions, or possibly of ulceration. As to the diagnosis of cancer in this way, much has been said. One authority recommends the use of a soft rubber tube with sharp-edged openings. The manipulation of this in the stomach may detach portions of the tumor, which can then be examined under the microscope. However desirable such examinations may be, the method recommended for securing the specimen is fraught with more danger than one is justified in incurring for the benefit to be attained. The practice should be mentioned only to be condemned. Of course, if particles of tumor are accidentally detached during lavage, advantage may be taken of the circumstance. *Thrush* may sometimes form in the stomach, in which case the vomited matter may contain large masses of the fungus of that disease.

It is now in order to study the various symptoms pertaining to the stomach, and which bring the patient to the physician.

1. **Flatulence.**—Here we have a symptom of more than passing importance, owing not only to the frequency with which it is encountered, but also because of the discomfort which it occasions the patient. In the clinical study of flatulence, it is of importance that its relation to time of eating and character of food, the character of the gases eructed, and the associated symptoms, are carefully considered. Cases of flatulence may be divided into two classes:

(*a*) Those dependent upon *decomposition of food*, in which case the gas will have both taste and odor.

(*b*) Those *dependent upon nervous causes*, in which event the gas is odorless and tasteless. The origin of the flatus in these cases has afforded a subject for considerable discussion. In many instances it is undoubtedly due to the air swallowed with the saliva. Careful observation of the patient in many of these enables us to attest to this fact, notwithstanding strenuous denials on the part of the sufferer. In others there can be no question that it is dependent upon an atonic condition of the gastric walls, the result of a negative pressure. It is said, also, that air may appear in the stomach as the result of secretion from gastric vessels. Such a hypothesis seems to be very doubtful.

In this connection, a reference to a personal experience may not be unprofitable. In an interview with my friend, Dr. D. W. Shoemaker, that gentleman remarked that he could draw a large quantity of air into his stomach and then eructate it ; and he made his assertion good. With a little experience I found that I could do the same thing, finally without giving any external evidence of the act. Indeed, frequent repetition seemed to make the act an involuntary one. The distress occasioned by this drawing-in of air was unquestionably uncomfortable in the highest degree. I can well see how a neurotic subject in the act of sighing can unconsciously bring about the same result, and thus start the vicious circle of phenomena embracing the clinical history of nervous dyspepsia.

The distinction between neurotic flatulence and gastric fermentation is a very important question in practice, as dietetic measures can rarely if ever benefit the former, while absolutely necessary in the latter. The quantity of gas eructed in the gastric neuroses is remarkably large—so large, indeed, that it is inconceivable that it can be derived from any other source than the swallowing of air. This condition may constitute the main symptom, if not the only one present. Careful study of some of these cases demonstrates that the gas eructed comes from the œsophagus. Most of the patients are found to be neurotic or hysterical.

It is said that flatulence may result from the regurgitation of pancreatic juice through relaxation of the pylorus, caused by the decomposition of the carbonates in the pancreatic secretion by the acid contents of the stomach. It hardly seems possible that flatulence from this cause can be of any great degree.

Objectively, flatulence may be manifest by great distention of the stomach.

Flatulence from fermentation is very commonly attended by cramp-like pains, which are relieved after eructations.

When flatulence from any cause is great and long-continued, it may disturb the action of the heart by its pressure, causing palpitation, distress, anxiety, and even fainting. Hence it not infrequently gives rise to fears of the existence of organic heart disease. Remembering all this, the physician must not ignore its importance in patients with muscularly weak hearts, in whom the consequences of flatulence may prove to be quite serious.

The flatulence due to regurgitation of pancreatic juice comes on four or five hours after eating.

2. **Vomiting.**—There is probably no symptom possessed of a wider clinical significance than vomiting, arising as it does not only from direct disease of the stomach, but also from reflex or nervous influences proceed-

ing from practically every organ of the body. The act itself originates in a sudden compression of the stomach brought about by spasmodic contraction of the abdominal muscles and diaphragm, in association with relaxation of the cardiac orifice of the stomach. Each of these factors, existing alone, is incapable of giving rise to vomiting. If the cardiac sphincter maintains its tone while the diaphragm and abdominal muscles are spasmodically contracted, retching only results; while, if the sphincter is relaxed and there is no expulsive force, the sense of nausea only exists.

The widespread origin of vomiting is readily appreciated when one bears in mind, first, the fact that there exists a vomiting centre in the brain capable of exciting the act, though all other parts are healthy; and, secondly, that the stomach receives its nerve-supply from the pneumogastric nerves, which also distribute filaments to many other viscera as well. In the clinical study of cases having vomiting as a symptom, therefore, one can reach a correct conclusion only after studying not only the special symptoms relating to the vomiting itself, but also all other associated clinical phenomena.

A study of the special symptoms relating to the vomiting itself demands attention to the following points:

a. The alleged cause of the vomiting.
b. Its relation in time to the taking of food.
c. The effect of the vomiting on associated symptoms.
d. The character of the vomited matters.
e. The associated symptoms.

(*a*) **The Alleged Cause of the Vomiting.**—This, as a rule, affords but little diagnostic information, excepting in those instances in which the partaking of some particular kind of food seems to have been the foundation of the trouble. Thus, in toxic gastritis the relation between the vomiting and the food that produced it is quite clear, especially in cases in which a number of persons have been attacked. Again, vomiting occurring after violent emotions or shock leaves no doubt whatever as to its nature.

(*b*) **The Relation in Time to the Taking of Food.**—Vomiting may take place immediately after eating, from various causes. Thus, in case of stricture at the cardiac orifice, as in malignant disease, food is ejected almost at once and unaltered, and not mixed with gastric fluids. Very similar vomiting (?) is observed in hysterical subjects, but the associated symptoms prevent any diagnostic error. The violent ejection of food immediately after it enters the stomach indicates a highly-irritable condition of that organ, and is found almost invariably in severe, acute toxic gastritis. Even the lightest food is vomited. Under such circumstances the stomach demands rest.

Vomiting shortly after taking food, that is, in the course of a half-hour or thereabouts, is not uncommon in chronic gastritis. In ulcer, the time of vomiting is about the same as in that of chronic gastric catarrh. In cancer, the time of vomiting is extremely variable, although, as a rule, it takes place later than in either chronic catarrh or ulcer. Usually, too, it does not take place until the disease is tolerably well advanced. It seems to be favored especially by stenosis at the pyloric orifice. When the latter condition has produced dilatation of the stomach, then the vomiting recurs at long intervals, and is of large quantities of food.

Certain conditions excite vomiting at special times of the day. Thus, the morning vomiting of pregnant women is well known to every physician. It is worthy of note that in many instances of this disorder the taking of food settles the stomach. Sometimes the vomiting is greatly relieved by mental and physical rest. The vomiting of those addicted to long-continued excessive indulgence in alcoholic liquors, although dependent upon catarrh of the stomach, is generally most marked in the morning, and is not infrequently relieved by the taking of liquor.

(*c*) **The Effect of the Vomiting on the Associated Symptoms.**—In many gastric affections vomiting brings relief to the suffering. In the acute forms of gastritis it must be regarded as a conservative process. The stomach is inflamed, and the presence of any food therein acts as a foreign body, and serves to intensify the local lesions. The patient experiences relief of his local and general distress as soon as the organ is emptied. Vomiting under such circumstances is not to be interfered with, as it is curative.

Among the chronic gastric affections in which the local symptoms are relieved when the stomach is empty are ulcer and cancer. The relief, however, is much greater in the former than in the latter. Indeed, it is so great as to afford a valuable diagnostic sign.

In chronic catarrhal gastritis, the relief of symptoms, epigastric distress, and pain, is only partial after vomiting.

(*d*) **The Character of the Vomited Matters.**—The materials ejected from the stomach include food, mucus, pus, blood, bile, and fæcal matter.

When food is ejected unaltered, it is safe to assume either that it has not entered the stomach, or that the gastric function is in abeyance. In the latter case it will be found mixed with other substances, which are discoverable by chemical analysis or inspection. The appearance of food ejected some time after eating affords information as to the activity of the digestive process. It has no diagnostic value as indicating the disease giving rise to it, as food is the substance which one expects to see in the vomit, and it must occur in all cases of disease accompanied by vomiting.

When mucus is observed in the vomited matters it is important to determine its source, as it may have been mixed with the gastric contents in the œsophagus, throat, or mouth. Associated symptoms will usually enable us to decide this point. The presence of mucus is recognized by the tenacious, stringy character of that substance. Chemically it may be determined as follows: The suspected substance is mixed with cold water and gently shaken. After settling a short time, the supernatant fluid is poured off and liquor potassæ added. This dissolves the mucus. Acetic acid is next added, and this precipitates the mucus, which is not dissolved by an excess of the acid, herein differing from syntonin. Mucous vomiting is observed in all affections of the stomach in which there is catarrhal inflammation, but more frequently in acute and chronic gastric catarrh than in any other.

Watery vomit is an important symptom of cholera morbus and cholera Asiatica. In both of these diseases the exudate from the gastric walls is remarkably large.

Purulent vomit is very rare, and can only occur in the presence of a suppurating lesion, as abscess, phlegmonous and membranous gastritis, and carcinoma affecting the lesser curvature and fundus. In the latter case, it may be possible to obtain from one-half to one ounce of pus from the non-digesting organ. When pus is found in larger quantities than this, it is a fair presumption that it has come from an abscess which has ruptured into the stomach,—as abscess of the liver, suppurative pancreatitis, or subdiaphragmatic abscess. In some cases pus may be present in such small quantities that a microscopic examination is necessary for its detection.

The vomiting of blood is a most important and alarming symptom of frequent occurrence. It indicates most surely the existence of a solution of continuity of the gastric mucous membrane, hence can occur only in ulcer, cancer, superficial abrasions, and diseases attended by degeneration of the gastric blood-vessels leading to their rupture. It is important at the outset to state definitely the symptoms by which vomiting of blood (hæmatemesis) is differentiated from the coughing up of blood (hæmoptysis).

In hæmatemesis the blood is brought up by the act of vomiting; it is usually dark in color, and mixed with food. Owing to its admixture with the gastric contents it is acid in reaction, and the patient gives a history of epigastric pain and disorder of digestion.

In hæmoptysis the blood is brought up by the act of coughing; it is nearly always bright in color, frothy, and mixed with mucus; its reaction is alkaline. Usually the history of the case shows the presence of pulmonary symptoms. Still it is not unusual for hæmoptysis to be the initial symptom of pulmonary disease.

Profuse vomiting of blood is especially liable to occur in cases of gastric ulcer, in some cases being in such large quantities as to cause collapse. When the bleeding is active it is soon ejected, and it is brought up unaltered.

When, on the other hand, it escapes by prolonged oozing, it is acted upon in part, at least, by the gastric juices, by reason of remaining some time in the stomach. In such cases it is dark, or even brown in color. Sometimes hæmorrhage takes place into the stomach, in cases of ulcer, without exciting vomiting. It is then associated with all the constitutional symptoms usually associated with profuse losses of blood. Later the blood appears in the stools, which present a black, tarry appearance.

Cancer of the stomach is rarely attended by the profuse bleeding found in ulcer. Moreover, the bleeding takes place slowly. Hence it is that the vomited matters present an appearance that has not inaptly been compared to "coffee-grounds."

Cirrhosis of the liver by reason of the production of portal congestion may lead to dilatation and subsequent rupture of the gastric and œsophageal blood-vessels.

Certain constitutional diseases, by reason of the production of alterations in the composition of the blood and degenerations of the vessels, give rise to hæmatemesis. Notable in this respect are scurvy, small-pox, yellow fever, the essential anæmias, purpura, and acute yellow atrophy of the liver.

Renal disease, especially chronic interstitial nephritis, may, though rarely, give rise to gastric hæmorrhage for the same reason that it may produce apoplexy, namely, the production of sclerosis of the blood-vessels and increased arterial tension.

Cases of hæmatemesis have been reported as the result of vicarious menstruation and neurotic influences. Cases of this character must be rare. In all of them, however, the escape of blood can only take place in the presence of a lesion which permits the escape of the blood from the vessels. There must be something more than mere neurotic conditions. There is nothing occult in hæmorrhage. It is a stern reality.

Sometimes blood is vomited when the source of the bleeding is elsewhere than the stomach. Thus, in epistaxis and œsophageal varices, it may be swallowed. Malingerers sometimes endeavor to create a sensation by the drinking of the blood of an animal, and then exciting vomiting. In duodenal hæmorrhage there may be regurgitation of blood into the stomach.

In cases of hæmatemesis in which the blood has undergone changes, chemical tests are sometimes necessary for its detection. After filtering the vomited matters, the filtrate is evaporated on a watch-glass. A small portion of the dried residue is then placed upon a glass slide with some finely-pulverized salt. One or two drops of glacial acetic acid are then added, and the mixture is put under a cover-glass. The slide is then heated gently over an alcohol flame. When dry, a few drops of water are allowed to flow under the cover-glass, and this dissolves the salt. If blood is present, the microscope will discover the brown rhombic crystals of the hydrochlorate of hæmin.

The guaiacum test is also available, though not free from error, as bile, saliva and starch may give rise to a reaction similar to that of blood. Two or three drops of tincture of guaiacum are added to a portion of the gastric contents in a test-tube. Next ozonic (ozonized) ether is added, but is not permitted to mix with the fluid already in the tube. Where the two liquids join there will appear, if blood is present, a blue line. In order to discriminate between blood and bile, a portion of gastric contents may be treated with nitroso-nitric acid (Gmelin's test), and if bile is present, the color-reaction will appear.

The presence of bile in vomited matters tells only of the fact that there has been regurgitation through the pylorus. Such a condition may arise when the pylorus is relaxed or there is stenosis of the small intestine below the orifice of the common bile-duct. In violent and prolonged vomiting, it is rare indeed that some regurgitation of bile into the stomach does not take place; hence the later vomitings will be found mixed with that substance. It is recognized by its yellow or green color and bitter taste. If there is any doubt about the nature of the suspected substance, Gmelin's test for bile may be applied.

The term "*fæcal vomiting*" is usually applied to the presence of a fæcal odor to the vomited materials, although in many instances the actual contents of the intestines are present. As a rule, the recognition of this symptom is not difficult, as the odor is diagnostic. If there is any doubt, a portion of the vomited matters should be distilled after the addition of a small quantity of phosphoric acid, and the distillate examined chemically for the presence of skatol, phenol, and indol. When the fæcal matter is derived from the small intestine, it will be found to contain bile acids and pigment and abundance of fat, as determined by microscopic and chemical examination. In the majority of cases fæcal vomiting is indicative of intestinal obstruction, especially of ileus. The presence of actually-formed fæces in the vomited materials is suggestive of a fistulous communication between the stomach and some portion of the intestine, usually of the colon. In the single case of this character seen by me, the trouble was primarily a gastric carcinoma, which had ulcerated, formed adhesions between the stomach and colon, and produced a fistulous communication between these organs.

The *animal parasites* found in the vomit are with rare exceptions those having the intestines as their favorite habitat. The ascaris lumbricoides, oxyuris vermicularis, and anchylostoma duodenale have frequently been observed, though with rarity relative to their frequency in the intestinal contents. Very exceptionally, portions of tape-worm, trichinæ, and the booklets of the echinococcus, have been discovered in the vomited substances.

(*c*) *The associated symptoms* are of the utmost value in determining the cause of vomiting. It has been stated in a preceding page that vomiting may originate not only from constitutional states, but also from disease in almost every organ of the body. To be more specific, this symptom may arise from general toxæmias, diseases of the central nervous system, heart, lungs, liver, kidneys, stomach, intestines, uterus, ovaries, and bladder.

The general toxæmias which may excite vomiting include the various infectious diseases, uræmia, etc. In their recognition we are obliged to rely exclusively, with but few exceptions, upon the associated symptoms, as there is little or nothing in the character of the stomach-symptoms to suggest their origin.

Cerebral vomiting is notably of projectile character. This symptom does not occur with equal readiness in all brain diseases. Its frequency is greater in young subjects. It is especially common in meningitis, constituting one of the four cardinal symptoms of that disease, the remaining three being fever, headache and delirium. It is commonly observed in tumors of the brain, being practically always present when the lesion is at the base. It is probably more common in abscess than in tumor. As one of the initial symptoms of apoplectic seizures, its importance is greatly underestimated. It is not at all unusual for vomiting to precede the cerebral symptoms by periods ranging from half an hour to a whole day. It presents the usual features of cerebral vomiting, being easy, and unattended with nausea or retching. Violent efforts at vomiting may, of course, be the exciting cause of cerebral hæmorrhage ; but in such instances the violent retching, long-continued, producing cerebral congestion, is the important factor, and there is abundant information to demonstrate that the stomach is the primary seat of disease.

The vomiting of uræmia is usually of morning occurrence, and is associated with nausea and depression. Physical examination discovers high pulse-tension, accentuated aortic second sound, and cardiac hypertrophy.

In respect to the vomiting of the infectious diseases, it should be borne in mind that it is not infrequent for this to constitute the initial symptom of the illness, so that at first it may be thought that a simple stomach trouble exists. Especially does this remark apply to the illnesses of children, in whom vomiting occurs from any slight causes. In many instances the severity of the vomiting is an index of the severity of the disease, as in yellow fever and small-pox.

A most interesting and often overlooked cause of vomiting is disease of the post-nasal space. Hawking and clearing of the throat is often constant in such cases, and the repeated efforts make the pharynx unduly irritable. Such a condition, it is well known, may readily excite reflex contractions of the stomach. The vomiting of post-nasal disease is very apt to follow attempts at clearing the throat, and is especially apt to be worse in the morning. Inspection of the pharynx and fauces usually makes the diagnosis clear.

The vomiting attendant upon lung diseases is usually produced by the violent spasmodic coughing. It is very seldom indeed that pulmonary disorders excite vomiting by simple reflex action without the intervention of coughing. The importance of coughing in this respect must not be overlooked, for many times it has happened that patients with pulmonary tuberculosis have been led to consult the physician, not because of the respiratory symptoms, but because of the annoyance produced by vomiting. Indeed, in many of them the cough seems to have been overlooked entirely until their attention has been directed to it by the medical man, and the nature of the case demonstrated by the physical signs of pulmonary lesions.

Of especial clinical importance is the recognition of the cause of the vomiting which ushers in certain acute abdominal disorders, and which calls for prompt action in order to save life. Thus, in appendicitis the characteristic abominal pain is sometimes associated with vomiting from the first. Indeed, the distress from the latter symptom may overshadow the pain to such an extent as to lead the patient to ignore its presence when describing his case to his physician.

Vomiting may constitute the only symptom appealing to the patient in case of incarcerated hernia, the tumor being so small as to be undiscoverable by expert examination, or having been of such long existence as to no longer obtrude itself upon the patient. In general peritonitis vomiting is an early and persistent symptom, and it is nearly always present in localized peritonitis from any cause. It is commonly present in intestinal obstruction. Its development here depends very much upon the location of the lesion. The higher the obstruction is situated, the earlier does the vomiting appear, and the more persistent it is.

The vomiting dependent upon pancreatic disorders is associated with sudden pain in the epigastrium, followed by collapse when the lesion is an acute pancreatitis. Cancer of the pancreas is associated with vomiting in conjunction with paroxysmal colicky pain and persistent jaundice.

3. **Thirst.**—Thirst is of more clinical value as indicating constitutional states than actual pathological conditions. By this term is meant the sensations which convey to the patient the demand of the system for water. These sensations are referred to the back of the throat. They can be relieved only when water has been introduced into the system, and not by simply moistening the parts. It is found in all conditions in which the body has lost fluids by dehydration or evaporation. Hence it is one of the prominent symptoms in diabetes, the large quantity of water being demanded to replace that lost in the polyuria. This symptom when persistent, and in association with emaciation, calls strongly for examination of the urine for sugar. In diseases characterized by profuse sweating, severe watery diarrhœa, and great loss of blood, thirst is a prominent symptom.

It is a natural demand on the part of nature, and should be gratified. In cholera Asiatica and cholera morbus, the severe serous vomiting acts as a producer of thirst in addition to the watery discharge accompanying these disorders, by reason of the profuse secretion of serum from the gastric mucous membrane. Incessant vomiting from gastric disease *per se* produces thirst because water does not remain sufficiently long in contact with the gastric mucous membrane to be absorbed.

Thirst, when present in chronic catarrhal gastritis, is especially marked towards evening. This affords a valuable differential symptom between chronic gastric catarrh and simple atonic dyspepsia.

Thirst is most distressing following the vomiting of acute peritonitis.

Thirst is a prominent symptom of all fevers, indicating a desire for cooling drinks. It is caused by the pyrexia *per se.*

Absence of thirst or demands for water occur in many conditions with blunted sensorium, *e.g.*, typhoid fever. This is important to bear in mind, as such patients should have water given them at regular intervals, whether they ask for it or not.

4. **Appetite.**—By appetite is meant the desire for food. As a result of disease, it may be lessened, increased or altered.

Increased appetite, or bulimy, occurs in convalescence from fevers and other acute disorders, after alcoholic beverages, in diabetes, in gastric irritation (neuroses), with relaxation of the pylorus, and in worms. The bulimia of convalescence is a demand on the part of nature for forced nutrition. It is therefore a conservative symptom. While catering to it in a measure, it must be remembered that there are conditions under which satisfying it is likely to do harm, as in a too early period of convalescence from typhoid fever. Diabetes is characterized by emaciation, and this loss of tissue likewise demands an increased quantity of food. In gastric neuroses the constant uncomfortable sensations demand the frequent taking of food, which brings relief for the time being in a large number of cases. In many of these, also, the impoverished nervous system requires an extra amount of food for its sustenance. In relaxed pylorus the large appetite has a very obvious cause. Food does not remain in the stomach long enough for that organ to act upon it. Hence the repeated demands for a new supply.

Loss of appetite is technically known as anorexia. It occurs in the course of all febrile affections, in acute and chronic diseases, especially those associated with diminished secretion, the taking of too highly nutritious food, and the abuse of alcohol.

The loss of appetite in fevers is the direct result of the diminished gastric secretion found whenever pyrexia is present. The stomach is not able to take care of the normal quantity or quality of food; hence the wisdom of keeping such patients on a spare and liquid diet. In acute and chronic catarrhal gastritis appetite is often wanting. Here, again, we have a wise provision on the part of nature, for the stomach is not able to take care of the normal quantity. Especially does this remark apply to cases in which the gastric secretion is altered. Those addicted to alcohol have notoriously bad appetites; their stomachs seem to require highly stimulating food for its satisfaction.

Alterations of appetite are known as "pica." Exceptional instances in which the perversions of appetite were remarkable have been reported. Under the morbid impulses, the patient eats of the most disgusting or indigestible articles. These cases are observed almost exclusively among pregnant and hysterical women, and among the idiots and the insane.

5. **Pain.**—Pain excited by disease of the stomach is situated characteristically in the epigastrium. Still, it must be remembered that other causes than those arising from the stomach itself may originate pain in this locality. Failure to recognize this fact has given rise to many mortifying diagnostic errors. For example, it may be

(*a*) **Neuralgic,** either from a simple intercostal neuritis or zona. I can well recall a case of epigastric pain in which I neglected to make an inspection of the parts, and diagnosed gastric disorder. The patient returning a few days later unimproved, an examination was made and revealed the presence of the vesicles of zona. Muscular rheumatism or myalgia may also occur here, as well as in numerous other portions of the body.

(*b*) **Localized peritonitis** may produce epigastric pain with sensitiveness. Under such circumstances there will be fever, probably extension of the pain and tenderness to surrounding portions of the abdomen, and a history of an illness likely to produce peritonitis in this locality, as gastric ulcer with perforation.

(*c*) **Affections of the pancreas** as a cause of pain in the epigastrium are especially liable to be overlooked—owing, probably, to their rarity. They will be duly considered when studying the pancreas.

(*d*) **Aneurysm of the aorta,** when the cause of epigastric pain, is rendered sufficiently obvious by its physical signs, and the extension of the pain along the course of the nerves irritated by the tumor.

(*e*) **Gall-stones** do not characteristically produce epigastric pain, but exceptionally the suffering is referred to this locality.

(*f*) In **caries of the vertebræ,** epigastric pain may occur when the disease involves the sixth or seventh dorsal vertebræ. This is an exceedingly important point to bear in mind, because this symptom constitutes the only symptom in many cases, and for months may be treated as an obstinate dyspepsia.

(*g*) **Various Disorders of the Stomach.**—This particular branch of the subject of epigastric pain concerns us more fully than any of the others enumerated.

(*h*) **Addison's Disease.**—The great prominence of the asthenia and the pigmentation of the skin led to the neglect of other symptoms common in this affection. Evidences of gastro-intestinal irritation are frequent, and include nausea, vomiting, and epigastric, hypochondriac and lumbar pain. These symptoms are due to the irritation of the sensory fibres in the neighborhood of the suprarenal capsules. Sometimes the pains are paroxysmal, and are associated with vomiting and nausea. They not infrequently resemble the gastric crises of locomotor ataxia. They may be associated with considerable rigidity of the abdominal muscles.

(*i*) **Acute Yellow Atrophy of the Liver.**—The epigastric pains in this disease are part of the gastro-intestinal disturbances which are almost always present in this disease.

(*j*) **Pericarditis.**—The pains of pericarditis are situated in practically all cases in the præcordial region. Nevertheless, a reflex pain may be exceptionally referred to the epigastrium. Such cases are very uncommon, and their nature can be recognized only after a thorough physical examination of the patient.

(*k*) **Strain or Rheumatism of the Abdominal Muscles.**—The diagnosis of epigastric pain from this cause is rarely difficult. The history of a strain, or of previous attacks of rheumatism, the superficial character of the pain, its aggravation on motion and on straining at stool, sufficiently indicate its nature.

The **disorders of the stomach associated with pain** are:

a. Acute catarrhal and toxic gastritis.
b. Gastralgia.
c. Gastric ulcer.
d. Cancer of the stomach.
e. Crises.
f. Hyperchlorhydria.
g. Atony of the stomach.
h. Gout of the stomach.
i. Chronic gastric catarrh.

(*a*) The pains of **acute catarrhal** and **toxic gastritis** vary only in degree. In the latter, the suffering is usually intense, and is greatly aggravated by the persistent efforts at vomiting, and only in less degree by the taking of any food. In catarrhal gastritis, pain may range all the way from a slight discomfort to severe pain. In chronic catarrhal gastritis it is very rare that there is anything more prominent than a discomfort or uncomfortable feeling after eating. Possibly the pressure of the clothing may be annoying. Pain is an important feature of the gastritis from irritant poisons, as arsenic and corrosive sublimate.

(*b*) **Gastralgia.**—The pain in gastralgia comes on in paroxysms, and is characteristically cramping and gnawing. It doubles the patient up; indeed, relief is afforded by that position. It often comes on when the stomach is empty, and may be relieved by the taking of food. In some cases it shows a remarkable tendency to recur periodically. The suffering sometimes shows itself on the constitutional condition of the patient, who is thrown into a collapse, with slow pulse, cold sweat, etc.

True gastralgia is seldom seen. Hence it behooves the practitioner to exercise the greatest care lest he confound it with one of the more commonly observed painful disorders of the stomach. The history of the case is of the greatest importance. Before one is justified in diagnosing gastralgia it must be possible to exclude the possibility of any other disease, because the failure to diagnose some of these may lose a life by reason of not applying proper medical or surgical treatment, while the failure to recognize gastralgia is not likely to do much harm.

(*c*) In **gastric ulcer** the pain, as a rule, bears a very close relation to the taking of food, being always aggravated after eating. It is greatly relieved after the stomach has been emptied by vomiting. There is often a localized sensitiveness. A not unusual situation for pain in this disease is in the back, to the left of the spinal column, and midway between the midscapular region and lumbar vertebræ.

(*d*) The **pains of gastric cancer** are usually aggravated by the taking of food, but not as characteristically so as in the case of ulcer. The pain, moreover, is constant, not exhibiting the great relief with an empty stomach observed in ulcer.

(*e*) **Gastric Crises.**—These occur almost exclusively in locomotor ataxia. They constitute one of the early phenomena of some cases. The pains are characterized by their great severity and their resistance to treatment. They have been known to recur for years before their true nature was recognized. They have been diagnosed as any of the painful disorders of the abdomen. Their diagnosis rests upon the symptoms of locomotor ataxia, as absent knee-jerks, Argyll-Robertson pupils, static ataxia, lightning pains in other portions of the body, syphilitic history, etc.

(*f*) **Hyperchlorhydria.**—The physical conditions in hyperchlorhydria sufficiently indicate the features of the attendant pain. It is generally aggravated when the stomach is empty, and it is relieved by the taking of food or by the exhibition of alkaline remedies, as the bicarbonate of soda.

(*g*) **Atony of the Stomach.**—Actual pain is not a prominent or important feature of gastric atony in any of its stages. It usually amounts to but a sense of pressure or distention after the taking of food, the patient feeling tolerably comfortable when the stomach is empty. The time after the taking of food during which the pain persists is a fair index of the severity of the case. In mild cases, all uncomfortable subjective sensations disappear within an hour, while in the more advanced cases the sensations of fullness and pressure may continue from one meal to another. In these, the morbid sensations are likely to be more marked after breakfast than any other meals.

(*h*) **Gout of the Stomach.**—The occurrence of gastric pain alternating with gouty swelling of joints is well known. Aside from this, we not infrequently observe cases of irregular gout in which the brunt of the disease is borne by the stomach, and in which the principal symptom is a gastralgia. A review of the histories of these cases will nearly always discover that the patient has experienced the unequivocal joint-symptoms of gout.

(*i*) **Chronic Gastric Catarrh.**—Pain is not a common feature of chronic gastric catarrh. Hence we are inclined to negative a diagnosis of that disease in the presence of this symptom. True as this is, we should not be too positive in our conclusions, for exceptions, though rare, do occur. Under such circumstances, the diagnosis of chronic gastric catarrh should never be made until one is able to exclude the possibility of the presence of all of the more common causes of epigastric pain.

This seems to be the proper place to refer to those numerous uncomfortable sensations related to digestion, and which cannot be regarded as actually painful, distressing though they are to the patient. In the vast majority of instances, these sensations are the expression of a pure neurosis. Sometimes they seem to be due to hyperchlorhydria, in which case they are greatly relieved by the taking of alkalis, as the bicarbonate of soda, or by a diet of meat. It is of the greatest importance to determine their neurotic character from a therapeutic standpoint, for it will never do to treat them by hard and fast dietetic rules.

6. **Pyrosis, or Water-Brash.**—Commonly known as "heartburn." This may exist as an apparently idiopathic affection, or it may be symptomatic of gastric disorder. It is characterized by a burning pain or distress in the epigastrium, which is relieved on the ejection of a small quantity of clear fluid, usually not over two or three ounces in quantity. It comes up without vomiting. The source of this fluid has been a subject of considerable discussion, it having been attributed in turn to the saliva, the œsophagus, the stomach, and the duodenum. As a rule, the fluid is tasteless. Still, it has been known to be quite acid. It seems to be more common in neurotic women than in others. It also constitutes one of the minor ills of pregnancy.

Schematic Presentation of the Symptomatology of Diseases of the Stomach.

When disturbance of the digestive functions suggests that we have before us a case of gastric disorder, it is well to keep in one's mind the list of diseases of which that organ is capable. This is an easy matter, for they are not numerous. At the same time, one must also not forget that the stomach may be disordered secondarily to lesions of other portions of the body, the primary disease being discoverable only by physical examination.

The various disorders of the stomach may be tabulated as follows:

1 Acute gastric catarrh.
2. Acute toxic gastritis.
3. Acute phlegmonous gastritis.
4. Ulcer of the stomach.
5. Cancer of the stomach.
6. Dilatation of the stomach.
7. Gastroptosis.
8. Achylia gastrica.
9. Isochymia.
10. Hyperchlorhydria.
11. The various neuroses, which include neuroses of
    (a) Motion.
    (b) Sensation.
    (c) Secretion.

1. Acute Gastric Catarrh; Acute Catarrhal Gastritis; Acute Catarrhal Dyspepsia.

This very common disorder presents a varied symptomatology, dependent upon the severity of the exciting cause and the predisposition of the individual. It is nearly always brought on by indiscretions in diet. The personal factor is, however, a very important one, for some persons are liable to catarrhal indigestion after very slight indiscretions. In the milder cases the symptoms may consist of some slight epigastric discomfort, and possibly also some nausea and vomiting. The more severe may be characterized by sharp epigastric pain, nausea, vomiting, foul breath, epigastric tenderness, vertigo, constipation or diarrhœa, and constitutional disturbances shown by the presence of profound prostration and fever. As a rule, the pain disappears promptly after giving the stomach rest. The vomited matters at first consist of imperfectly digested food only, but as the process continues, mucus and bile will be ejected. As a rule, however, the emptying of the stomach at once quiets it, so that empty retching, which is such an important feature of toxic gastritis and cholera morbus, is a very rare phenomenon of catarrhal inflammation. Examination of the vomited matters is rarely necessary, or even of importance, in establishing a diagnosis. The fact that the disease is of acute onset, and that the vomited matters contain mucus, evident on simple inspection, is all-sufficient. The fever is rarely persistent or of high grade in the catarrhal dyspepsia of adults, usually disappearing as soon as the stomach is thoroughly emptied. In young children, on the other hand, the febrile movement constitutes a most important clinical feature. It comes on suddenly, not infrequently in the midst of apparently good health, and may lead to a suspicion that one of the infectious diseases is about to appear. Scarlatina is, however, the disease above all others which may be suspected at this stage ; but in that disease we have a knowledge of epidemic prevalence, the disproportionately rapid pulse, and possibly, also, evidence of disturbed digestive function.

The greatest danger of diagnostic error lies in the frequency with which numerous diseases, as croupous pneumonia, the acute infections, meningitis, peritonitis, appendicitis, etc., exhibit vomiting and other gastric symptoms, in association with fever, as their initial phenomena. It seems to me, however, that the attendant difficulties in these cases are more imaginary than real, for careful systematic examination of patients as a routine method of practice will prevent one falling into such diagnostic pitfalls.

2. Acute Toxic Gastritis.

This is practically a high grade of gastric inflammation produced by some poison introduced from without. Especially active, in this respect, are such mineral poisons as arsenic and corrosive sublimate, and the ptomaines —especially those of spoiled sausage and fish, canned goods, and ice-cream. The symptoms are characterized by their suddenness of onset and their violence. Severe pain is probably the initial symptom, followed very shortly by vomiting, which continues until the stomach is thoroughly emptied. Even then, however, the irritability of the stomach does not subside in many instances, as shown by the long continuance of empty retching. The retching and vomiting, by their violence, react on the inflammatory condition, intensifying it, and increasing the pain. Up to the time that the vomited matters contain food or its remnants, it is wisdom to do nothing to restrain it. It is different, however, when the empty retching occurs. The mucous membrane of the stomach is found to be remarkably irritable, as shown by the readiness with which the blandest food-stuffs or drinks re-excite vomiting. In some of the cases the bowels likewise participate in the inflammatory process, as shown by severe diarrhœa, with profuse watery stools, griping pains, and flatulence.

3. Phlegmonous Gastritis; Gastritis Phlegmonosa Purulenta.

This may occur as a primary disorder, or it may be metastatic. In any event, it is an exceedingly rare affection. There can be no question of the fact that it is due to infection. There may or may not be premonitory dyspeptic symptoms. The first phenomenon of the disease itself is severe pain in the epigastrium attended by a violent burning in the stomach, great thirst, dry tongue, complete loss of appetite, and high fever, which usually follows the characteristic septic type. Systemic affection being marked, the pulse is weak; prostration is generally profound. The diagnosis is almost impossible during life, as its symptoms find a close counterpart in other forms of acute gastritis. It may be suspected with good reason in the presence of the septic type of fever, especially when associated with repeated rigors, and when there is profound prostration, and especially if a possible source of infection is known. The disease is an exceedingly fatal one, life being maintained for but from four to seven days.

4. Chronic Gastric Catarrh; Chronic Catarrhal Dyspepsia; Gastritis Glandularis Chronica.

Hardly a disease affecting the human body has as varied an etiology as this. Its causes may be either local or general. The local include persistent disregard of dietetic precautions, of which, in my opinion, improper methods of feeding and cooking, excessive indulgence in alcohol, and overfeeding, are of more importance than the usual character of the food itself. Such causes must operate over a considerable length of time, however, before they produce chronic gastric catarrh. In the meantime, they are provocative of numerous acute seizures, which, coming closer and closer together, are finally perpetuated in the chronic disease. Constitutional diseases of all kinds vitiate the gastric functions as they do the functions of other organs, and thus they lay the foundation of chronic catarrh of the stomach. Chronic heart and pulmonary diseases lead to venous congestions, and in no organ any more prominently than in the stomach; hence catarrhal dyspepsia may be a complication of these affections. It must be remembered that chronic catarrh constitutes an associated lesion of all the chronic organic diseases of the stomach, and impresses some of its features on those of the primary disease.

The symptoms are of gradual onset. Discomfort is experienced after the taking of food—sometimes only after particular articles, and sometimes after everything. Food is not digested thoroughly, and hence decomposes and gives rise to flatulence, the eructed gas tasting of the food or having a putrid or disagreeable flavor. There is usually more or less general disturbance, manifested particularly by nervous symptoms. These include mental depression, general nervousness, drowsiness, especially after eating, headache, disturbed restless sleep, and general torpor. In the more advanced cases there may be nausea and vomiting, the ejected matters including, in addition to more or less digested food, quantities of mucus. Good judgment is necessary to differentiate chronic gastric catarrh from the nervous affections which may simulate it.

When the illness has continued for a long time, the mucous membrane of the stomach undergoes secondary changes; that is, it loses in strength, or its glandular structures atrophy, producing dilatation of the stomach in the former instance, and so-called "atrophy of the stomach" in the latter.

Examination of the gastric contents in the simple forms of catarrh discovers an abundance of mucus, and at the same time a diminution in the quantity of free hydrochloric acid and pepsin.

5. Gastric Ulcer.

The etiology of this affection is not fully understood. It is known that it is comparatively common in women who are exposed to hot fires, as in-cooking, and also in anæmic young women. Some claim that it is the result of the action of a normal or hyperacid gastric juice upon diseased portions of the mucous membrane, which are incapable of resisting the chemical action of gastric secretion.

The typical picture of gastric ulcer includes localized epigastric pain with tenderness, greatly aggravated by the taking of food, and relieved by vomiting and vomiting of blood, often in large quantities. These various features have already been described in preceding pages (*vide* p. 223), and need not be reviewed here. Atypical cases, however, are occasionally observed, and call for the greatest judgment for their recognition. Thus, there are instances in which the ulcer has given rise to no symptoms whatever, its presence not being suspected until perforation or death from other causes. In others it has occasioned the symptoms of gastric catarrh only, and in still others hæmorrhage of sudden appearance has been the sole clinical condition.

Emaciation and anæmia, while often present, are never extreme. The disease may exist over a long term of years. It is not uncommon for the patient to make a complete recovery under treatment and then relapse. As regards these relapses, it does not necessarily follow that gastric trouble succeeding a healed ulcer is a recurrence of the old disorder. Cancerous changes sometimes, though rarely, take place in the old cicatrix; or the contraction of the cicatrix interferes with the mechanical functions of the stomach; or the original trouble may have excited inflammation of the parietal peritoneum, and this, in turn, has caused adhesion to adjacent viscera. It becomes an important matter, therefore, to be able to differentiate ulcer from the effects of ulcers,—a problem that is often of not very easy solution.

6. Dilatation of the Stomach; Gastrectasia.

This is always a secondary condition. Its pathological causes are atony of the gastric walls or obstruction at the pyloric orifice. The former is a consequence of gastric catarrh, especially in those who habitually overdistend the stomach. Obstruction of the pylorus may be produced by cancerous disease in this locality, benign tumors, and cicatricial formation from healed ulcerations. Exceptionally, there occurs an acute dilatation of the stomach known as paralytic distention. Prominence of the upper portion of the abdomen appears suddenly and physical examination discovers the stomach to be enlarged and tympanitic. The upward pressure of the dilated organ interferes sadly with the functions of the heart. The weakened condition of the gastric walls prevents the expulsion of flatus. When vomiting occurs, it is severe.

Chronic dilatation of the stomach is attended by all the usual concomitants of cartarrhal gastritis. There is a peculiarity, however, about the vomiting. The patient vomits at long intervals, but the quantity ejected each time is unusually great. The vomited matters, moreover, give abundant evidence of their retention in the stomach. They are yeasty in appearance, and under the microscope are found to contain sarcinæ, torulæ, and other products of fermentation.

7. Carcinoma of the Stomach.

The etiology of cancer of the stomach is that of malignant growths in other localities. It is believed that traumatism and long-continued abuse of the stomach is an important predisposing factor. Like ulcer, this affection presents remarkable symptomatic types. The typical case has its beginning at or beyond middle life. At first there is experienced a loss of appetite without apparent cause, which persists for several weeks without yielding to treatment. Then pain is experienced, and this gradually increases in severity, until finally it becomes agonizing. As already stated, this pain is aggravated after taking food, but this relation is by no means as marked as in the case of ulcer. Hæmatemesis also enters into the symptomatology of the disease, but the bleeding is rarely free. Usually, too, the blood is retained for some time in the stomach, and undergoes alterations; hence when it is vomited it presents a "coffee-ground" color. The presence of a tumor, however, is the only reliable symptom upon which a diagnosis of cancer of the stomach can be based. Unfortunately this reliable sign does not appear until the case has advanced so far as to make curative surgical treatment out of the question. The same remark applies to the cachexia and emaciation which sooner or later appear in all cases. Examination of the gastric contents shows a diminution of free hydrochloric acid, with the presence of free lactic acid and the Oppler-Boas bacillus. Metastasis takes place to other organ tissues, notably to the liver and mesenteric glands. However desirable an early diagnosis of cancer of the stomach may be, we will never attain our object if we rely upon any one special symptom. An early positive conclusion cannot be reached except by taking the case as a whole. Any one point helping to the opinion is susceptible of varied interpretations when taken alone, but, studied in conjunction with the associated symptoms, will make early accuracy probable.

8. Gastroptosis.

By this term is meant a downward displacement of the stomach. It is usually associated with similar displacement of the intestines (Glenard's disease) and kidneys. The subjective symptoms are those of digestive disturbances in general, and are not characteristic of any one in particular. The diagnosis must be based entirely upon the physical signs, which show the stomach to occupy a lower than the normal position in the abdomen.

9. Achylia Gastrica.

By this term is meant an absence of the secretion of gastric juice. This condition is a rare one, and was first described by Einhorn, of New York. The symptomatology of this affection appears at first sight to be anomalous. Notwithstanding the entire absence of digestive fluid, there are cases presenting no symptoms whatever, the digestive functions being carried on entirely by the intestines. In a second class of cases gastric symptoms are present, but are associated with intestinal symptoms as well; and in a still third class the intestinal symptoms constitute the entire clinical picture of the case, the bowel disturbance arising from the extra work thrown upon the parts by the failure of the stomach to prepare food properly for intestinal digestion. The diagnosis rests upon the analysis of the gastric contents and the absence of symptoms indicative of cancer, and other conditions presenting a similar analysis.

10. Isochymia.

This is a condition characterized by the retention of food in the stomach, even in the fasting state. It is practically a motor insufficiency of the stomach. It may appear as either an acute or as a chronic condition. As an acute disorder it is the result of inflammatory changes which, for the time being, suspend the motor-power of the stomach, and probably exists in some degree in most cases of acute gastric catarrh. In some cases, also, it may be due to spasmodic contraction of the pylorus. Chronic isochymia is nearly always due to pyloric obstruction, as from cancer, ulcer, etc. Considerable fermentation in the stomach nearly always exists, and is responsible for many of the symptoms. Appetite is poor. There is considerable oppression. Thirst is prominent. There are eructations of gas having offensive odor and taste. Vomiting is present, and includes the expulsion of large quantities of material, a portion of which represents food taken into the stomach, possibly the day before. Auscultation of the stomach will detect the swashing and sizzling sounds of fermentation.

11. Hyperchlorhydria or Excessive Formation of Free Hydrochloric Acid.

This is a condition occurring for the most part in persons who have been subjected to worry and excessive mental application. It may be also produced by habitual indulgence in highly-seasoned dishes, alcoholic liquors, and ice-water. The onset of the disease is gradual. In the early stages the patient experiences an uncomfortable sensation in the stomach, coming on about two or three hours after eating. This pain disappears as soon as some nourishment is taken, apparently because the introduction of food gives the hyperacid contents of the stomach something to act upon. It also disappears after taking the bicarbonate of soda,—which, of course, neutralizes the free acid. The epigastrium is usually found sensitive on pressure. Examination of the gastric contents gives an increased total acidity ; there is no impairment of the motor-function. The general condition of the patient, notwithstanding the severity of his dyspeptic symptoms, is most excellent. The bowels are generally constipated. The conditions which are liable to be confounded with hyperchlorhydria include gastric ulcer, hypersecretion, and biliary colic, from all of which they may be differentiated by the analysis of the gastric contents.

12. The Neuroses of the Stomach.

As already stated, the neuroses of the stomach may be divided into the (a) motor, (b) sensory, and (c) secretory neuroses.

(*a*) **Motor Neuroses.**—These include : i. Cardiospasmus ; ii. Eructation ; iii. Regurgitation ; iv. Rumination ; v. Nervous vomiting ; vi. Pneumoatosis ; vii. Akinesis ventriculi ; viii. Hyperkinesis ventriculi ; xi. Peristaltic restlessness ; x. Incontinence of the pylorus ; xi. Pylorospasmus ; xii. Atony of the stomach.

i. *Cardiospasmus ; Spasm of the Cardia.*—In this condition the patient has no difficulty in deglutition at first ; but after swallowing a few mouthfuls there appears a feeling of distress, due to the retention of food in the œsophagus. If nothing is done in the way of manipulations to relieve these morbid sensations, the food is regurgitated, and relief is obtained. The sufferer soon learns, however, to make pressure upon the lower portion of the thorax while holding his breath, and this forces the food into the stomach. He is then able to partake of more food, when the same set of phenomena appear. Quite naturally, in chronic cases, the œsophagus undergoes dilatation, and extreme instances have been reported in which that tube was capable of containing as much as 300 c.c. These symptoms indicate indubitably the existence of obstruction at the cardia. That such obstruction is not organic is demonstrated by the fact that a sound passes into the stomach without resistance. The dilatation of the œsophagus may be diagnosed by passing the sound as far as but not through the cardia, when the retained food can be withdrawn unaltered.

ii. *Eructation.*—The exact nature of nervous eructation is a matter of doubt. It is currently believed that the air is swallowed. The paroxysms of belching come on at irregular intervals, sometimes every few minutes. The expelled gas is tasteless and odorless. The trouble comes on while the patient is asleep. This condition occurs almost exclusively in nervous and hysterical subjects.

iii. *Regurgitation.*—By regurgitation is meant a condition in which food or drink is regurgitated shortly after swallowing. It is believed to be due to relaxation of the cardiac orifice of the stomach. While the trouble is very obstinate, it does not seriously impair the patient's health, as a rule.

iv. *Rumination* is characterized by regurgitation of food, which is chewed over again and re-swallowed. It occurs with especial frequency among the insane and idiots. It is very obstinate to treatment.

v. *Nervous vomiting* at times presents the greatest difficulties in diagnosis. For its recognition we depend upon the absence of signs and symptoms indicative of organic diseases, the presence of the neuropathic constitution, and the fact that the patient's general condition does not suffer in proportion to the extent of the vomiting. While the vomiting is liable to occur after meals, there is no special kind of food which seems to excite it. It may follow upon the ingestion of the simplest articles, and be absent after substances well-known to be indigestible.

vi. *Pneumoatosis* is a condition in which the stomach is distended with gas, and is believed to originate in spasmodic contraction of both cardiac and pyloric orifices. It is associated with symptoms arising from the pressure of the distended stomach.

vii. *Akinesis ventriculi* is a condition characterized by defective gastric motility. It may occur as part of the clinical picture of organic lesions, as ulcer, or it may be an idiopathic neurosis. It can only be recognized by the gastrograph. (Einhorn.)

viii. *Hyperkinesis ventriculi* is a condition characterized by morbid activity of the stomach walls.

ix. *Peristaltic restlessness* is likewise a form of excessive gastric motility, but differing from hyperanakinesis in that the peristaltic movements are readily visible, moving as waves from left to right. In many instances it is a pure neurosis, but in the majority it is dependent upon some obstruction at the pyloric orifice, the symptoms of which will also be present.

x. *Incontinence of the pylorus* may be dependent upon organic lesions at this end of the stomach, or upon gastric atony. The data upon which we are to depend for its recognition do not seem to be well-defined. Most authorities are not willing to accept an early emptying the stomach after a meal as satisfactory evidence, as they contend that such a state is brought about by excessive activity of gastric motility. The symptoms upon which most reliance has been placed is a free regurgitation of intestinal contents into the stomach, as demonstrated by the introduction of the tube after a test-meal.

xi. *Pylorospasmus* is, in the majority of instances, a reflex from organic disease, as ulcer. The symptoms resemble those of organic obstruction at the pyloric orifice. It sometimes occurs in conjunction with hyperchlorhydria and hypersecretion. The region over the pyloric orifice is found tender or painful to palpation, but too much reliance must not be placed upon this symptom.

xii. *Atony of the stomach, or atonic dyspepsia,* is a condition dependent upon a weakness of the muscular apparatus of the stomach. It may be one of the features associated with organic gastric diseases, or it may be an expression of a neurotic constitution, or due to a general constitutional ill-health, as in phthisis. The more prominent symptoms include a sense of fullness or weight, coming on immediately after eating; flatulence, with eructation of tasteless gas; poor appetite, headache, and constipation. As important diagnostic symptoms may be mentioned a splashing sound, produced by palpation over the epigastrium, and the abnormally long time which food is retained in the stomach after eating (six hours). In many cases it will be found that, as the stomach is distended with larger and larger quantities of water, the greater curvature of the organ descends lower and lower.

(*b*) **Sensory Neuroses.**—The principal sensory neuroses of the stomach are as follows: i. Bulimia; ii. Polyphagia; iii. Akoria; iv. Nervous anorexia; v. Gastric idiosyncrasies; vi. Abnormal sensations; vii. Hyperæsthesia of the stomach; viii. Gastralgia.

i. *Bulimia,* also known as *hyperorexia,* is a condition of abnormal hunger. It may be either primary or secondary. In the former case it is a pure neurosis, and may be constant or paroxysmal; in the latter it is symptomatic of ulcer, helminthiasis, cancer, hysteria, and other affections. The craving for food when the desire comes, which it may do without warning, is irresistible. Failure to take food causes untold distress. In the majority of cases, only a small quantity of food is required to give relief.

ii. *Polyphagia* is an excessive craving for food which can only be satisfied by the taking of incredible quantities. Like bulimia, it may be primary or secondary. The latter class of cases is observed in conjunction with cancer of the pancreas or spleen, diabetes, and some cases of tumor of the brain. As examples of the extremes to which these patients may go to satiate their cravings may be quoted the case of Rosenthal, that of a woman who ate at one meal an entire goose and a big portion of bread.

iii. *Akoria* is an absence of the sensation of satiety. The patient never knows when he has partaken of a sufficient quantity of food. It presents the same symptomatic value as bulimia and polyphagia.

iv. *Nervous anorexia* is a very common gastric neurosis. It is characterized by almost entire absence of desire for food, without explainable cause for the loss of appetite. The patient is usually anæmic or weak, and yet these conditions are not as marked as one would expect from the small quantity of food taken. In contradistinction to cases of symptomatic anorexia, the absence of appetite does not occasion the patient any alarm. Care must be observed not to confound nervous anorexia with the loss of appetite observed in connection with the incipiency of serious organic diseases, as pulmonary tuberculosis.

v. *Gastric idiosyncrasies* are probably more common than physicians suspect, as patients, knowing their peculiarities and the poor prospects of cure, say little or nothing about the trouble. The substances concerning which the idiosyncrasy is manifested include especially strawberries, eggs, shell-fish and fish. In any individual case, the symptoms resulting from the dietetic transgression are always the same for that particular case.

vi. *Abnormal sensations* in great variety may be reported by neurotic subjects. These include sensations of heat and cold, sense of fullness even when the stomach is empty, pulsations, cramps, nausea, etc.

vii. *Hyperæsthesia of the stomach* is manifested usually by sensations of fullness or distention after meals. In some cases the discomfort amounts to actual pain or distress. In other cases the gastric irritability is so great as to occasion vomiting. Emaciation is not a prominent feature of the latter cases, because, as a rule, the patient vomits only a portion of his food, the other portion being retained and assimilated. The diagnosis is based upon our ability to exclude the organic affections liable to be associated with gastric hyperæsthesia and the presence of the neuropathic constitution.

viii. *Gastralgia* has been sufficiently described when speaking of the diagnostic value of epigastric pain. It is only necessary, in this place, to call to mind the fact that gastralgia may exist as a pure neurosis; from gastric disease; from spinal-cord affections; from constitutional weakness, as chlorosis; and reflex, from disease of other viscera.

(*c*) **The Secretory Neuroses.**—The only gastric secretory neurosis of importance is the so-called *nervous dyspepsia*. This, in the majority of cases, forms a part of a general neurasthenia or hysteria. It is not uncommon to speak of it as gastric neurasthenia when it occurs as the sole manifestation of the neurotic state. It may, however, constitute part of the clinical features of some of the organic dyspepsias, thus leading to diagnostic errors. In some cases it is a direct result of one of the acute infectious diseases, especially of influenza. Excessive indulgence in alcohol, tobacco, or sexual intercourse, may also produce it.

The symptoms of nervous dyspepsia include capricious appetite, discomfort or distress in the epigastrium after eating, eructations of tasteless and odorless gas, general weakness or prostration, vertigo, and, above everything else, the most gloomy forebodings or depressed mental state. The bowels are nearly always constipated. Notwithstanding the alleged severity of the suffering of the patient, outward appearances of malnutrition are noteworthy for their absence. While the symptoms are alleged to be due to the ingestion of food, it is evident that no particular food disagrees with any regularity. When the patient gets away from the trials and tribulations of his daily duties, all of the symptoms disappear, almost as if by magic.

The Intestines.

Examination of the Intestines.—The physical examination of intestinal changes or conditions involves the practice of

1. Inspection.
2. Palpation.
3. Percussion.
4. Auscultation.
5. Inflation of the intestines with gas.
6. Test-lavage of the intestines.

1. **Inspection.**—The examination of the abdomen by inspection should be practised first with the patient in the standing position, after which it should be continued while he is lying down. The reason for this advice is very apparent. When the patient is standing, partial bulgings as well as uniform enlargements become more prominent because of the greater weight borne by the abdominal walls; and, moreover, it not infrequently happens that these disappear with the patient in the recumbent position.

Perfect illumination of the parts to be examined is an absolutely essential prerequisite to accuracy of observation. It is only by attention to this point that nice distinctions as to differences in elevation can be determined.

The patient should be placed upon a couch or table with head and legs extended, and the abdominal muscles should be as thoroughly relaxed as possible. It is important that observations should be made both during quiet or ordinary and deep respiration.

First the physician should direct his attention to the skin over the abdomen, the special changes which he may expect to discover being striæ and venous enlargements.

Abdominal striæ show themselves as scar-like markings over the abdominal walls. They are generally parallel and in groups, and are due to cicatricial tissue following breaks in the subcutaneous cellular tissue and the cutis, brought about by abdominal distention; *e.g.*, in ascites and pregnancy. They may also be produced by large accumulations of fat in the abdominal walls. When of recent formation they are of a reddish hue; when old, an almost dead white. They persist after the cause of the increased intra-abdominal pressure has been relieved.

Enlargements of the abdominal veins may be produced by increased intra-abdominal pressure (ascites and tumors) interfering with the return flow of blood from the lower extremities, portal obstruction (cirrhosis of the liver), thrombosis of the iliac veins or inferior vena cava, mediastinal tumors, and obstruction in the superior vena cava. The venous enlargements may be but moderate, and exhibit themselves as unduly large veins coursing over the abdominal walls. On the other hand, they may be extreme, and present a large bunch of tortuous veins centreing about the umbilicus (the *caput medusæ*). When the obstruction is in the inferior vena cava, the veins of the lower extremities will also be enlarged, and not infrequently there is an associated œdema.

In all cases of enlargement of the abdominal veins in which the origin of the difficulty is not clear, an attempt should be made to determine the direction in which the blood flows through the dilated vessel. For example, there may sometimes be seen an enlarged vein running upwards in the mid-axillary line. When it is dependent upon obstruction of the inferior vena cava, or of the portal vein, the blood-current is upwards; when, on the other hand, it originates in obstruction of the superior vena cava, the flow is downwards.

When the veins enlarged are limited to the pubic region, the causative obstruction must be below the liver.

Visible enlargement of the epigastric arteries indicates possible obstruction of the iliac arteries or aorta.

All information from the inspection of the abdominal surface having been obtained, attention is next directed to the shape or contour of the abdomen, especial attention being paid to local or general enlargements. The importance of inspection in the recognition of intra-abdominal growths has been greatly underestimated, mainly because the examination is not conducted under suitable light conditions. Boas says: "*Even small new growths are much more readily detected by careful inspection with a good light (a very important point) than they are through palpation.*" It is scarcely necessary to remind the reader that the evidence afforded by inspection as to intra-abdominal enlargements is proportionately reliable, as the abdominal walls are thin and emaciated.

Uniform enlargement of the abdomen may be due to

a. Accumulations of fat in the abdominal walls and around the abdominal organs.

b. Meterorism or tympanites.

c. Ascites.

When generalized, abdominal enlargement is due to accumulation of fat; other portions of the body, as the chest, arms and legs, will show a like condition. Occasionally one meets with individuals in whom all the fatty accumulation seems to be deposited in the abdominal walls, the balance of the body being *relatively* thin.

The diagnostic relations of meteorism or accumulation of gas within the intestinal coils will be considered in the succeeding portion of this chapter, under the head of *intestinal flatulence.* At this time I will refer only to the differentiation of gas confined within the intestines (intestinal meteorism) and gaseous accumulations within the peritoneal cavity. The latter condition can only exist as a result of some lesion capable of producing perforation of a gas-containing organ, *e.g.*, the stomach and intestines. Consequently the previous history of the case—*e.g.*, typhoid fever, traumatism, or intestinal or gastric ulcer—constitutes an important point in determining a conclusion. Under certain circumstances, inspection offers some suggestions. Immediately after the occurrence of the accident (perforation), the abdominal enlargement is uniform. Later, when peritonitis supervenes, and with it an exudation of serum, the enlargement, though still uniform, varies with the position of the body. The exudate, being heavy, falls to the most dependent parts. With the patient lying on his back, it occupies the posterior portion of the abdominal cavity, the lateral walls of which bulge unduly as compared with the anterior abdominal walls. Still later, when the distention becomes extreme, the enlargement again becomes uniform, independently of any position the patient may assume.

Circumscribed or irregular enlargement of the abdomen may be due to

a. Tumors.

b. Herniæ.

c. Localized accumulations of flatus.

Diminution in size or retraction of the abdomen may be due to

a. Extreme emaciation.

b. Colic of lead-poisoning.

c. Basilar meningitis.

d. Spastic contractions of single intestinal loops.

Increase of intestinal peristaltic activity may be discovered by inspection when the abdominal walls are thin. This condition may occur in the early stages of lead-colic, and in occlusion of the intestines. It sometimes occurs as a visceral neurosis, usually in association with a similar condition of the stomach.

2. **Palpation.**—By palpation of the abdomen, as applied to the discovery of abnormal conditions of the intestines, we may determine

a. The presence or absence of sensitive points.
b. The presence or absence of localized muscular rigidities.
c. The condition of special portions of the intestines.
d. The presence or absence of tumors.
e. Œdema, emphysema, and corpulence of the abdominal walls.
f. Splashing and friction vibrations.
g. Herniæ.

The method of practising abdominal palpation has already been described in the section devoted to the consideration of conditions and symptoms of the stomach. When palpating the abdomen as a whole, it is wise to accustom one's self to some particular routine, in order that no region be neglected. Naturally, one directs his attention to the condition of the abdominal walls, and thus becomes aware of the presence or absence of œdema, emphysema, excessive accumulations of fat, or tumors, although the experienced will have already recognized these conditions by inspection. Next, when palpating for intra-abdominal lesions or conditions, it is a good plan to begin in the epigastrium, and then, in turn, direct attention to the hypochondria; to the region of the small intestines and about the umbilicus; to the large intestines, beginning with the cæcum or appendix (McBurney's point); and then follow the course of the ascending, the transverse and the descending colon, and the sigmoid flexure. Finally, the various canals through which hernia may protrude should be palpated.

(*a*) **The Presence or Absence of Sensitive Points.**—When palpation is practised for the purpose of discovering generalized or localized sensitiveness, pressure should always be gentle, for pain, or at least discomfort, may be produced by rough manipulation. Moreover, roughness may readily do harm in the presence of actual lesions. Care should also be exercised when examining nervous, apprehensive individuals, who may imagine the presence of pain or soreness when neither exists. When there is a suspicion of such a state of affairs, the examination should be repeated after a few minutes' rest, and the result compared with the first observation. One must not permit himself to be deceived by the hyperæsthesia so commonly observed in hysterical or neurasthenic subjects.

Sensitiveness over the McBurney point (midway between the anterior superior spinous process of the ilium and the umbilicus) is almost certainly indicative of appendicitis.

Pain and sensitiveness along the descending colon and over the sigmoid flexure is found in acute dysentery.

Circumscribed pain over the duodenum is symptomatic of ulcer in that locality. The sensitiveness must be persistent. It is not felt much beyond its focus of greatest intensity.

Multiple circumscribed sensitive points are found in catarrh of the small intestines complicated by follicular ulcers, which ulcerations are, as a rule, of tubercular origin.

Pressure sensitiveness in the left iliac region may be observed in chronic sigmoiditis.

Pain over the entire abdomen, with sensitiveness to the slightest touch, is observed in general peritonitis.

(*b*) **The Presence or Absence of Localized Muscular Rigidities.**—Little or no importance can be attached to a general muscular rigidity during palpation, as it is commonly dependent upon the patient's nervousness. Nevertheless, there are cases in which associated symptoms indicate that it is dependent upon general tenderness. Local muscular rigidities possess the same diagnostic significance as localized tenderness to palpation; indeed, they may be regarded as adding to the certainty of the conclusions.

(*c*) **The Condition of Special Portions of the Intestines.**—Just how far observations in this line are reliable is at present uncertain. Obrastow, as quoted by Boas, claims to have been able to locate the cæcum in 51.47 per cent. of the men and in 58 per cent. of the women he had examined; the transverse colon in 23 per cent., and the sigmoid flexure in 65 per cent. of all of his cases. The confidence of the practitioner in these results must be weakened by the failure of Obrastow to confirm them by post-mortem examinations. There can be no doubt, however, that the sigmoid flexure is the most readily-accessible portion of the large intestine to palpation, and that the cæcum is a close second. The recognition of the sigmoid flexure by palpation is made easier by reason of its being filled with fæcal masses. It has been suggested that the physician induce an artificial coprostasis, in order to discover thereby the existence of displacements of the intestinal coils. When palpating the cæcum or the lower portion of the ileum, one feels the sensation as of a thin, pasty mass under the fingers, as well as the localized gurgling so common in this situation.

(*d*) **The Presence or Absence of Tumors.**—The tumors which may involve the intestines include carcinoma, sarcomata, tuberculomata, and very exceptionally the different varieties of benign tumors. They become increasingly frequent as we reach the lower portions of the intestinal tract. Indeed, it might be said that, if we except cancer of the rectum and sigmoid flexure, malignant disease of the intestinal tract is so rare as to be almost a clinical curiosity. Osler mentions it only in conjunction with the etiology of intestinal obstruction. From a clinical standpoint, this is wise, because carcinoma of the intestines grows in an annular direction, and so impairs the intestinal lumen. It is not uncommon for the intestines above to be dilated, sometimes enormously so. The movability of intestinal neoplasms will depend entirely upon the portions of the canal they involve.

The duodenum being fixed, tumors in this situation have a definite location. On the other hand, the sigmoid flexure, the coils of the small intestines and the transverse colon, having long mesenteries, will permit of considerable movement of any tumor connected with these parts.

Tumors of the peritoneum are practically always secondary to similar growths elsewhere. The tumors are nearly always multiple, scattered over the peritoneal surface, and are associated with some degree of peritonitis and effusion. As Osler has graphically stated it, " The clinical history is that of chronic ascites with progressive emaciation." The greatest difficulty in diagnosis seems to be in the differentiation of tuberculosis and carcinoma. The latter is especially liable to occur in persons at or beyond middle life, while tuberculosis finds its most favorable soil in childhood and youth. The nodular tumors of carcinoma are much larger than those of tuberculosis.

Perhaps the most mortifying mistakes concerning the diagnosis of intestinal tumors are found in cases of coproliths (fæcal tumors). In the majority of instances this error may be entirely obviated if the observer will but take the precaution to have the bowels thoroughly evacuated prior to making his examination. These fæcal tumors are generally of doughy consistency and retain the impressions of the fingers. When they are situated in an intestinal diverticulum, or when perforated by a central canal, purgatives and other measures for emptying the intestinal tract may prove valueless for their removal. In this case their diagnosis may prove a very difficult matter, if not actually impossible.

Boas refers to Gersuny's " adhesive sign " as evidence confirming the suspicion of an intestinal tumor. " If the finger be very firmly pressed upon the fæcal mass, the intestinal mucous membrane will become adherent to the viscous fæces, becoming free again when the pressure is discontinued. Gersuny states that we can feel the mucous membrane loosening itself, and that such a sensation is characteristic of a fæcal tumor." Boas, however, has not been able to convince himself of the presence of this adhesive sign, and still regards the impressibility of the fæcal tumor as its most characteristic symptom.

3. **Percussion.**—Percussion offers but little help in the physical examination of intestinal conditions. The percussion-note, it is true, presents variations in tone and pitch over various coils, but this fact does not seem to be of much practical importance, as yet, from a diagnostic standpoint. Over large tumors, one of course will discover percussion dullness ; but in this case the nature of the lesion has already been determined by palpation and inspection. Perhaps the most valuable use to which percussion over the intestines may be applied is in the differentiation of intestinal meteorism from free gas in the peritoneal cavity. In the former the percussion-note, as stated, will vary over different intestinal coils ; while in the latter the tympanitic reso-

nance will be exactly the same all over, and the hepatic and splenic percussion dullness is found to have disappeared.

4. **Auscultation.**—Of all the physical methods for examination of the intestines, auscultation presents the most limited sphere of utility. By it we may determine the activity of the gurgling sounds of peristalsis—a most important thing for the abdominal surgeon, who regards total absence of such sounds as suggestive of peritonitis.

5. **Inflation of the Intestines with Gas.**—The information to be obtained by this method includes the detection of strictures of the large intestine and their location, the more ready recognition of displacements of the large intestines by palpation and inspection, and the differential diagnosis of intestinal tumors from those of other abdominal organs.

Two methods of inflating the intestines with gas have been advocated. Ziemssen recommends that carbonic acid gas be employed. The other, which seems more generally applicable, is the introduction of atmospheric air by a double-bulb syringe attached to a long, soft-rubber tube. Comparatively little of the injected gas passes beyond the ileo-cæcal valve, thus causing the large intestines to become unduly prominent, and making them more evident to physical examination.

When intestinal stricture is present, the parts above the narrowing do not become distended.

After the intestines have been distended, deeply situated tumors of the organs disappear. Tumors of the intestines may become evident, especially when so situated as to become more closely coapted with the abdominal walls.

6. **Test-Lavage of the Colon.**—For applying this method of examination, a large Langdon tube about 80 cm. in length is required. By causing water to flow while it is being passed, folds of mucous membrane coming in contact with its point are pushed aside, and, with care, it may be introduced as far as the ileo-cæcal valve. Respecting the information to be obtained by the lavage, Hemmeter says: "Under normal conditions the experimental lavage of the colon shows very slight admixture of the water with fæcal particles, mucus, and epithelial detritus; but the water very often returns clear. In the various forms of colitis, however, mucus is abundant in the lavage water; and it also contains fragments of the colon mucosa, which are available for microscopic examination. On several occasions I have succeeded in finding fragments of neoplasms situated in the transverse colon in this manner. The method is available for the diagnosis of all the various types of colitis."

Symptoms arising from disease of the intestines result in one of three ways:

(*a*) From the particular pathological condition present, the symptoms being similar to those arising from that condition in any other portion of the body.

(*b*) From disturbance of function as a conducting tube, as part of the digestive apparatus, and as an organ of absorption.

(*c*) From constitutional disturbance.

Symptoms arising from the particular pathological process do not concern us at the present time, so we proceed to the study of those produced by disturbed function. It is the province of the intestines to pass the products of digestion along the tube. After the partially-digested food leaves the stomach and enters the intestines it undergoes further changes in its downward course. As certain parts become fully digested they are absorbed, while the remainder is passed onward and finally expelled by the rectum. This process is an exceedingly important one, impairment of which results in ill-health.

The activity of the bowels may be diminished, which we call constipation; or it may be increased, when the condition is known as diarrhœa.

1. **Constipation.**—As to what constitutes constipation is a legitimate question. Ordinarily, the healthy individual has one bowel movement daily. There are individuals who depart from this standard—some who have two evacuations daily, others who go to stool not oftener than once in two or three days. Age exerts an important influence on the frequency of stool. Thus, in infancy it is expected that there may be from two to four evacuations daily; while, in old age, movements of the bowels take place about every other day.

Important as is the subject of regular bowel movements, altogether too much attention has been directed to it by the laity and profession alike. The former are constantly dosing themselves with all sorts of purgative pills, placed on the market by not overly-scrupulous patent-medicine manufacturers, while the latter limit their therapeutic resources to such drugs as produce frequent bowel movements. On the other hand are those who, led by this one extreme, rush to the other, and neglect attention of the bowels to a degree detrimental to health. Between these two extremes there is a happy medium, which can be judged by experience only.

The causes of constipation can be divided into classes, namely:

a. The constitutional, and
b. The local.

(*a*) **The Constitutional Causes.**—These include:

i. Sluggishness of function.
ii. Sedentary habits.
iii. Irregularity in going to stool; hurrying at stool, etc.
iv. Frequent use of purgatives.
v. Excessive use of alcohol, tobacco, coffee, etc.
vi. Dietetic causes.
vii. Indigestion.
viii. Diseases of heart, stomach, etc.
ix. Dehydration.
x. Fever.

i. **Sluggishness of Function.**—Cases of constipation due to this cause are very common. The subjects are for the most part women whose general nutrition is poor. Their physical stamina is low. They are highly neurotic, and often anæmic. Menstruation is deficient, and urinary excretion is much below the normal standard. Treatment directed to the relief of the constipation does absolutely nothing towards bringing relief, and the same remark applies to the amelioration of the other symptoms. The condition seems to be a clinical entity relievable only by general constitutional measures.

These cases must not, however, be confounded with another class in which chlorosis or one of the forms of anæmia exists as the prime cause, for in them the relief of the anæmia adds tone to the intestinal muscular fibres and corrects the constipation.

In still another class, generally consisting of patients of a lymphatic temperament, and often dependent upon hereditary influences, the constipation seems to be part of a sluggishness of general functions; the patient is slow to think, move and act, generally.

ii. **Sedentary Habits.**—Active physical exercise in the open air is necessary to a high standard of health, and is an important means of keeping the bowels regular. Bookkeepers, bankers, students, and others whose occupations keep them within doors for long hours, are prone to constipation. Such persons may protest that they are actively engaged. However that may be, their activity is not of the kind conducive to the tissue-changes promoting regularity of bowel action.

iii. **Irregularity in Going to Stool.**—The individual parts of the system, as well as the individual himself, form habits. Persons who cultivate the habit of going to stool at a definite hour each day rarely suffer from constipation. *Per contra*, those who are careless in this respect must expect irregularity. There are those who, with the regularity of habit disturbed even for one day, will suffer from constipation in consequence. This is seen in travellers.

iv. **Frequent Use of Purgatives.**—It is very rare, indeed, that the frequent use of purgatives conduces to regularity of bowels. Usually the intestines become accustomed to the undue stimulation, and finally become unable to act without it. Then, too, it must be remembered that when the bowels are thoroughly purged, there remains no residual fæcal matter, and this is necessary for a natural movement. Hence a secondary constipation ensues. This, in turn, leads the patient to resort to another purge; and so the vicious cycle is maintained.

v. **Excessive Use of Alcohol, Tobacco, Coffee, etc.**—These induce constipation, as a rule, by producing indigestion, or by weakening the intestinal muscles. It is an interesting fact that tobacco and coffee may, when used with judgment, be employed for securing regularity of stool.

Tea and red wine produce constipation by reason of the astringent action of the tannin they contain.

vi. **Dietetic Causes.**—To a certain degree, dietetic causes of constipation are physiological. The foods leading to this result are those of a highly-nutritious quality, leaving but little waste for fæcal formation. Necessarily, then, the intestines have but little to work upon. If this habit is continued for a long time, the constipation becomes pathological. The foods of this class include milk, eggs, meat and meat extracts. Constipation may also arise from taking of too limited quantity of fluids.

Exceptionally, constipation may result from the taking of coarse, heavy food, which, by reason of the large bulk of its waste, so distends the intestinal walls as to greatly weaken their muscular power.

vii. **Indigestion.**—The constipation is part of the general inactivity of the gastro-intestinal tract. It also arises in part from the alterations in the composition and quantity of the digestive fluids. Conditions in which the biliary secretion is deficient are usually attended by inactivity of the bowels, because bile is a most powerful stimulus of intestinal peristalsis.

viii. **The heart,** when diseased, may interfere with the circulation of the intestines, as with that of the stomach, to disturb its functions.

ix. **Dehydration of the body** from any cause induces the absorption of water from the intestinal contents, thus leading to hardening of the fæces and constipation. Thus, from excessive sweating, whether from disease, hot weather, or immoderate exercise, constipation may result. Diabetes mellitus, by reason of the large quantity of urine excreted, produces the same result.

x. **Fevers** of all kinds are almost invariably associated with inactivity of the bowels, because of the diminution of gastro-intestinal secretions in diseases attended by this symptom. The small quantities of food administered to febrile cases also play an important part. The only exceptions to the statement that constipation is an attendant of fevers is found in those affections which have diarrhœa as a symptom.

2. **The local causes of constipation** include:

   i. Narrowing of the calibre of the intestines, as by displacement of an intestinal coil, the formation of cicatrices after ulcers, tumors, intussusception, etc.
   ii. Fæcal impaction.
   iii. Weakened muscular action of the abdominal walls, as in old age, pregnancy, obesity.
   iv. Painful affections of the abdominal walls, *e.g.*, rheumatism.
   v. Local conditions of the anus, as fissure, spasm, hæmorrhoids, and rectal ulcer.
   vi. Pelvic pain, as in ovaritis and pelvic suppuration.
   vii. Weakened intestinal walls from flatulent distention.

i. **Narrowing of the calibre of the intestines,** *i.e.*, intestinal obstruction. The pathological conditions which may give rise to acute intestinal obstruction are as follows:

Intussusception;
Volvulus;
Strangulation by peritoneal bands;
Strangulated hernia;
Gall-stones;
Intestinal calculus.

In addition to these, intestinal obstruction may be brought about gradually by

Cicatricial stenosis from a healed ulcer;
Intestinal tumor, usually malignant.

The symptoms of acute intestinal obstruction from any cause are well-defined. They are of sudden onset. Concisely stated, the cardinal symptoms of intestinal obstruction are sudden and severe pain, most obstinate constipation, collapse, and vomiting. The bowels cease to act notwithstanding medicinal and mechanical efforts employed to produce a stool. The obstruction may be so complete as to prevent the escape of flatus. Pain is usually present and is intense. It is generally described as of colicky or crampy character. Its location possesses little diagnostic significance, for, while it is frequently centred over the point of obstruction, it may have no relation whatever to the seat of the lesion. Usually the abdomen is sensitive, but at times the patient experiences relief by exerting firm pressure. Constitutional symptoms are early phenomena, the patient being collapsed, with pinched features, cold extremities, rapid, weak pulse, hurried respirations, and dry, brown tongue. The abdomen is early distended, from the accumulation of gas in the intestines above the point of obstruction. This symptom may serve as a guide in locating the lesion. Peristaltic movements of the bowels are increased above the obstruction. Hence, the higher the lesion the less generalized the peristalsis. In some cases these movements are visible through the abdominal walls. Vomiting occurs early, especially when the obstruction is high. At first it differs in no particular from the vomiting of gastric disease. After a few hours it presents a fæcal odor, and this is regarded as characteristic. True fæcal vomiting, however, does not take place until an advanced stage of the illness, and is to be regarded as evidence of complete obstruction. It does not occur, however, when the obstruction is in or above the jejunum.

While it is not always possible to determine the exact cause of the obstruction, nevertheless individual cases present important features possessed of considerable diagnostic value. Acute obstruction during early life is more frequently due to *intussusception* than to other causes. In this form of obstruction, moreover, pain is an early symptom, and is steady.

It may, however, subside suddenly. Vomiting does not occur so early, and is not generally severe. The constipation is not complete. Indeed, there is frequently a pseudo-diarrhœa, characterized by the discharge of blood and mucus. Tympanites is usually absent. Physical examination often discovers an oblong tumor early in the progress of the case. Later, when peritonitis sets in, this tumor is no longer palpable, because of the abdominal swelling. Sometimes a portion of the intestine protrudes through the anus, or may be palpated in the rectum.

Volvulus occurs more frequently in adult males. The lesion is usually situated in the larger bowel. Local tenderness is an early symptom. Vomiting is likewise late in its appearance, but when it does come on, it is persistent. Constipation is complete. Indeed, this symptom usually precedes those of obstruction by several days. Owing to the low position of the obstruction, tympanites is a prominent feature of most cases. Tenesmus, likewise, is present, though frequently absent. Peritonitis is a frequent and early complication.

Strangulation of the intestines by peritoneal bands and consequent obstruction is to be inferred when the symptoms of intestinal obstruction appear in persons who have previously suffered from attacks of peritonitis, appendicitis, and other diseases liable to produce peritoneal adhesions.

Several factors are of assistance in diagnosing the location of the obstruction. Pain, while not an infallible guide, is certainly a strongly suggestive one. When diffused over the entire abdomen, or of a generally colicky nature, it is of no assistance; but when sharply circumscribed, especially with localized tenderness, it may be regarded as of great value. Flatulence or tympanites is the more marked as the obstruction is the lower situated. Hence this symptom affords some information, though in a general way. Auscultation is of value as showing the portions of the intestines still in possession of their peristaltic power, the bowels being motionless below the seat of obstruction. Inspection and palpation may discover irregular distention of the bowels, and thus furnish valuable diagnostic suggestions.

Cicatricial stenosis from a healed ulcer is to be recognized by the history of the case. The majority of examples of this trouble are syphilitic in origin, hence the value of a specific history in forming an opinion. The lesion, moreover, is especially liable to appear in the sigmoid flexure of the colon. The patient usually gives a history of a long-continued painful affection of the bowels with discharge of pus, and possibly of blood in the stools, followed by a gradually increasing constipation. Palpation may discover a localized hardness at the seat of cicatricial contraction.

In *obstruction from tumor*, the primary trouble is usually recognized long before the symptoms of intestinal occlusion present themselves; hence diagnosis is rarely attended by any difficulty.

Intestinal obstruction from an enterolith is of the rarest occurrence. So far as the symptoms are concerned, their diagnosis presents insurmountable difficulties. Nevertheless, in one case seen by me many years ago, and in which the diagnosis was confirmed by Dr. W. B. Van Lennep, the calculus was palpable, and was so situated as to permit of ready removal had the unfortunate patient been able to overcome her unwarranted prejudice to surgery.

Intestinal obstruction from gall-stones must be even more infrequent than the cases produced by enteroliths. Gall-stones are rarely sufficiently large in size to obstruct the intestinal lumen. When such is the case, there has been a fistulous communication between the gall-bladder and intestines to permit of its escape. The diagnosis rests upon the previous history of the case, especially of evidence of disease of the gall-bladder.

ii. **Fæcal Impaction.**—This condition, under ordinary circumstances, should not be difficult of recognition, especially as the inactivity of the bowels permitting it must be of long continuance. The writer has seen several instances, however, in which grievous diagnostic errors were committed. Mistakes seem to have resulted from the fact that the irritation of the intestinal mucous membrane by the hard fæcal mass had provoked a pseudo-diarrhœa, the stools containing considerable mucus and but little fæcal matter. Added to this fact is the additional one of a general hardness of the lower bowel, resulting from the accumulated fæcal masses, and leading to the assumption that an abdominal tumor is present. Fæcal impaction rarely occurs, however, excepting in persons already debilitated by disease, excepting when the patient has been fed on improper food, seeds and husks accumulating in the lower bowel and becoming impacted.

iii. **Weakened muscular action of the abdominal walls,** as in old age, pregnancy, obesity, etc. The diagnosis of such cases is readily made by reason of the laxity of the abdominal parietes.

iv. **Painful affections of the abdominal walls,** *e.g.*, muscular rheumatism. Under such circumstances attempts at defecation result in abdominal pains manifestly aggravated by increased effort. Hence the patient not unnaturally restrains himself. The pain is a diffuse soreness, worse from any movement or pressure. Manipulation teaches that it is situated in the abdominal walls, and that it does not arise from within the abdomen.

v. **Local Conditions of the Anus, as Fissure, Spasm, Hæmorrhoids, Irritable or Inflamed Prostate, and Rectal Ulcer.**—Pain at the anus acts reflexly in restraining stool. More than that, the enforced inactivity of the bowels, by leading to intestinal accumulation, makes the pain at stool still greater, and so intensifies the constipation. Stools are apt to be hard, and excite bleeding. It is too common an error, under such circumstances, to diagnose hæmorrhoids, although it is true that these painful lesions are not an infrequent cause of this condition. Much more frequent, however, are small fissures, which also excite spasm of the anal sphincter.

vi. **Pelvic Pain.**—All forms of pelvic disorder characterized by inflammation and pain are attended by constipation, owing to the enforced rest of the parts. More than that, the constipation reacts upon the primary disorder and leads to passive congestions, thus establishing a vicious circle.

vii. **Weakened Intestinal Walls from Flatulent Distention and Other Causes.**—Long-continued stretching of muscular tissue from any cause leads to gradual deterioration of power. Hence, if the intestines are maintained in a state of distention for a prolonged period by habitual constipation or flatulence, weakness of their walls results.

A survey of the causes of constipation above described demonstrates that each of them acts in one of three ways :

1. By diminishing the amount of fæcal residue.
2. By modifying absorption of fluids from the intestines, and thus affecting the consistence of the fæces.
3. By diminishing intestinal peristalsis.

It is important, in the management of each case, that the particular function at fault is recognized, for in that way only can the trouble be corrected.

Symptoms Attendant upon Constipation.—Ordinarily, constipation produces a slight abdominal discomfort, with ineffectual urging to stool. The difficulty is corrected at once, and the trouble ends here. Exceptionally, however, very interesting results ensue, and sometimes fail of recognition. These may be enumerated as follows :

1. **Colic or Abdominal Pain.**—Paroxysms of abdominal pain of colicky character, recurring at more or less regular intervals, may occur from the intestinal irritation as the result of the retained fæcal masses.

2. **Intestinal inflammation** may follow in some few cases, because of the intensity of this irritation.

3. The inflammation, in its turn, especially if frequently repeated, brings about **weakening of the intestinal walls,** which yield to pressure. Or, the habitual distention of the bowels by fæcal masses may result in their dilatation.

4. **Venous dilatations** are sometimes produced by the pressure of the hardened fæces, the best illustrations of which are found in hæmorrhoids and varicocele.

5. **Uterine and ovarian disease** is a common attendant upon the constipation of women. The retained fæces act mechanically to produce pelvic congestion, and, if long continued, organic changes must ensue. This fact is overlooked with too great frequency. The tendency at the present

day is to attribute too much influence to septic infection and too little to the bowels.

6. **Left-sided sciatica** may be brought about by the local effect of a distended sigmoid flexure.

7. Exceptionally, **œdema of the feet** may be produced in the same way.

8. **Auto-Intoxication.**—The long retention of effete matter in the intestines leads to absorption of toxins, and, as a result, there follow numerous symptoms of auto-intoxication. These exist in great variety. Sometimes they are so distressing as to completely overshadow their cause. Generally they are very indefinite in character, though none the less serious in their effects.

2. **Diarrhœa.**—By diarrhœa is meant the frequent evacuation of stools possessed of a watery consistence. The stools themselves may consist of fluid fæces, mucus, serum, undigested food, and even of blood and pus. Associated with the diarrhœa are usually pain, flatulence, and sometimes tenesmus. The pain may be constant or paroxysmal. In some it is provoked by the irritation of the abnormal fæcal contents ; in others by the flatulent distention. As a rule, it is greatly relieved by stool or the escape of flatus. It is only in inflammatory cases that there is tenderness and steadiness of the pain. It is usually sharp and griping, sometimes described as lancinating. When inflammation is present it may be localized over the inflamed part, *e.g.*, the colon. The flatulence is, as a rule, not of a high grade. The flatus is more or less offensive. Tenesmus is always present when the stools are at all frequent. It is also a prominent symptom in cases in which there is inflammation or irritation of the rectum and anus. It must not be regarded as diagnostic of dysentery, a mistake only too common. In rectal complications of diarrhœa the tenesmus may be constant, and may even excite prolapsus of the anus. Dysentery is in reality an acute infectious disease having a well-defined course, and should not be confounded with diarrhœal troubles.

The constitutional disturbances associated with diarrhœa vary with the frequency and copiousness of the movements and the general condition of the patient. When the stools are frequent and profuse, there is usually more or less marked weakness, thirst, loss of appetite, scanty urination, and some fever,—usually, however, not more than two or three degrees. In the aged, diarrhœa is especially prostrating, and may prove to be a dangerous malady.

Diarrhœa may result from the following causes :

(*a*) *Direct irritation of the intestines*, as from over-feeding or gastric indigestion. Food enters the intestinal tract improperly prepared, and the intestinal juices are unable to take care of it. It then acts as an irritant, exciting abnormal peristalsis ; exudation of mucus and frequent loose stools follow. Recovery is prompt after the expulsion of the offending materials.

(*b*) *Purgatives and poisons.* The actions of certain drugs, as castor oil, aloes, croton oil, etc., in producing frequent liquid evacuations is too well known to need extensive elaboration here. Certain poisons, notably arsenic, mercury and antimony, and the ptomaines, include diarrhœa in their symptomatologies by reason of the high grade of gastro-enteritis they produce. In arsenical and ptomaine poisoning the diarrhœa is associated with vomiting, the slightest portion of liquid or food in the stomach exciting the act. Stools are profuse, watery and exhausting. Pain is a prominent feature.

(*c*) *The presence of too much bile.* Bile acts as a stimulant to the intestinal peristalsis. Hence, when present in excess, stools are abnormally frequent.

(*d*) *Hardened fæces.* These irritate the intestinal mucous membrane, exciting a flow of mucus and increased peristalsis. The stools, under such circumstances, are found to consist of scybalæ and mucus.

(*e*) *Irritation of parasites.* This is an unusual cause of diarrhœa. It can be diagnosed only by the recognition of the parasites in the stools.

(*f*) *Ruptured abscesses,* the stools containing pus which has made its appearance suddenly.

(*g*) *Defective hygiene,* breathing foul air, living in crowded rooms, etc.; a very common cause of diarrhœa in poor communities, jails, camps, etc. It is generally associated with bad food, uncleanliness, etc.; hence it is impossible to say just how much influence is exerted by each factor.

(*h*) *As a vicarious symptom.* This is especially observed in cases of impending uræmia. The stools become frequent, watery and profuse. Such a diarrhœa must be regarded as eliminative, supplementing the poor work of the damaged kidneys, and hence should not be interfered with.

I have also seen a very interesting phenomenon of diarrhœa substituting for the chill in intermittent fever, the cold stage being attended by profuse vomiting and diarrhœa, simulating in every way ordinary cholera morbus, but differing from that disease in its recurrence on alternate days at exactly the same hour.

(*i*) *Nervous disturbances.* This is a peculiarity of some people, who get diarrhœa under slight emotional causes, as fright or anxiety. As a rule the attacks are but short-lasting, disappearing on the subsidence of the excitement giving rise to it.

Under this heading may also be included some cases of diarrhœa occurring in hysterical subjects. As a rule, however, very strong evidence should be at hand, together with the absence of any other causative condition, before a diarrhœa of mysterious origin should be diagnosed as hysterical.

(*j*) *Rectal and anal irritation.* Fissures of the anus, ulcer, or stricture of the rectum, which, as already stated, usually produce constipation, sometimes give rise to the opposite result, and excite diarrhœa by either direct or reflex irritation.

(*k*) *Appendicitis.* Here, again, we have another disease in which diarrhœa is the unusual condition.

(*l*) *Constipation* may produce diarrhœa. This sounds paradoxical; and yet there is no fact in clinical medicine better recognized. The accumulation of hardened fæcal masses in the colon leads to irritation, with excessive secretion of mucus. The resulting stools consist of scybalæ covered with mucus, and sometimes containing blood.

(*m*) *Symptomatic diarrhœa* occurs in a number of diseases, as follows:

i. Acute intestinal catarrh.
ii. Acute membranous enteritis.
iii. Cholera infantum.
iv. Cholera morbus.
v. Cholera Asiatica.
vi. Dysentery.
vii. Typhoid fever.
viii. Septicæmia.
ix. Colitis.
x. Toxæmia.
xi. As part of the intestinal crises in locomotor ataxia.
xii. Membranous enteritis.

3. **Intestinal Hæmorrhage.**—Blood escaping by the anus presents different appearances, according to the portion of the intestinal canal from which it was derived and the rapidity with which it has escaped from the ruptured vessels. The nearer the source of hæmorrhage to the anus, the nearer does the appearance of the blood in the stools approach that of blood in color and the less intimately is it mixed with the fæces. The more remote from the anus the seat of hæmorrhage, the more likely is the bloody stool to present a tarry or pitch-like appearance. When the hæmorrhage is from the rectum or anus, the blood discharged is pure, and merely coats the fæcal masses.

Intestinal hæmorrhage may result from—

i. *Traumatism by foreign bodies.* The history of the case can only make the diagnosis certain.
ii. *Intestinal ulcerations,* as in typhoid fever, malignant disease, tuberculosis and syphilis.
iii. *Gouty kidney.* This cause is not sufficiently appreciated. Its mechanism is the same as in the case of hæmorrhages from the same cause occurring in other portions of the body, namely, arterial degeneration and increased intravascular pressure.

iv. *Blood changes*, as in scurvy, hæmophilia, pernicious anæmia, yellow fever, and many of the acute infectious diseases.

4. **Abdominal Pain.**—Abdominal pain, though most frequently due to lesions of intestinal origin, is by no means confined to the symptomatology of these affections. The intestinal affections of which it may be a symptom include the following:

a. Colic or enteralgia.
b. Intestinal indigestion.
c. Acute enteritis.
d. Chronic lead-poisoning.
e. Duodenal ulcer.
f. Malignant disease of the intestines.
g. Hernia.
h. Appendicitis.
i. Cholera morbus and Asiatica.
j. Dysentery.
k. Enteroptosis.
l. Fæcal impaction.
m. Intestinal obstruction.
n. Intestinal perforation.

(*a*) **Colic or Enteralgia.**—The pain in this condition is paroxysmal in its recurrence. It is described by the patient as griping in character. Its focus of greatest intensity is about the umbilicus. It is usually relieved by bending double or making firm pressure with the hands. It is often attended by constipation or flatulence, and disappears when these troubles are relieved. The diagnosis of colic should always be unsatisfactory to a physician, for the case cannot be said to have received an explanation unless the origin of the difficulty is also specified. The use of the term "enteralgia" as a simple nervous affection of the intestines should be condemned, for such cases are practically never observed, if we except the crises of locomotor ataxia, and the neuritic affection of the abdominal walls. Truly, the diagnosis "nervous pain" in the abdomen covers a multitude of mistaken diagnoses.

(*b*) **Intestinal Indigestion.**—The pain exhibits the colicky character. It disappears promptly as soon as the intestines are relieved of the offending materials.

(*c*) **Acute Enteritis.**—A variety of pains may be observed in acute enteritis. The most common is colicky in character, precedes an evacuation, and is relieved thereby. When the large intestines are involved, there may be associated tenesmus recti. Between the paroxysms of pain the patient experiences sensations of distress, discomfort, or actual soreness.

(*d*) **Chronic Lead-Poisoning.**—The patient is nearly always a worker in lead. The paroxysms of pain are severe, and there is associated retraction of the abdomen and most obstinate constipation. The presence of a blue line on the gums adds to the certainty of the diagnosis.

(*e*) **Duodenal Ulcer.**—This affection is characterized by a fixed pain situated somewhat below the location of the gall-bladder, on the right parasternal line. It has been described as exhibiting a burning or boring character radiating downwards or to the sides, and aggravated by lying on the right side. Its characteristic period of aggravation is about three or four hours after the taking of food. Chvostek has offered a suggestion by which the pain of duodenal ulcer may be differentiated from the corresponding lesion of the stomach. If pains about the epigastrium coming on some three hours after a meal are relieved by the taking of wine, it is suggested that the lesion is duodenal ulcer, because the wine causes a reflex closure of the pylorus. If, on the other hand, the ulceration is gastric, the pain is aggravated by the taking of wine. Boas accepts the above facts as stated, but explains them by the dilution of the hyperacid gastric juice by the food or liquid taken. As he expresses it, "Substances such as milk or egg-albumin, which have a strong tendency to combine with hydrochloric acid, should act better than wine."

(*f*) **Malignant Disease of the Intestines.**—Pain should be an important feature of all cases of malignant disease of the intestines. As in gastric carcinoma, it is constant, but is subject to periods of aggravation. The diagnosis, however, depends upon the obstinate character of the illness, the age of the patient, the cachectic condition of the patient, and, above all, upon the discovery of a tumor.

(*g*) **Hernia.**—The fact that hernia, though not strangulated and perfectly reducible, may produce abdominal pain, is not sufficiently recognized. Even in cases in which the supports in the rings or canals are weak, and as yet there is no protrusion, mild fugitive pains are not infrequent. They are not associated with tenderness, but are ofttimes aggravated or excited by sudden muscular exertion.

(*h*) **Appendicitis.**—The pain of appendicitis is probably possessed of a more fixed location than that of any other abdominal pain. Its point of maximum intensity is at the so-called McBurney point, *i.e.*, just above a point midway on a line drawn from the anterior superior spinous process of the ilium to the umbilicus. In very many cases the pain may be here, and not elsewhere. But it may happen that the abdominal pain is diffused, in which case the diagnosis is determined by the special sensitiveness at the McBurney point. This is an important point to bear in mind, for many cases of so-called colic are misunderstood because of failure to properly determine the location of maximum intensity of pains.

(*i*) **Cholera Morbus and Asiatica.**—The pain in these affections is that of enteritis, *i.e.*, griping and paroxysmal, with relief succeeding an evacuation of the bowels, with some soreness or discomfort, more or less constant.

(*j*) **Dysentery**, likewise, has constant and paroxysmal pains, but there is associated severe tenesmus recti.

(*k*) **Enteroptosis.**—Because of the secondary organic changes which may take place in the intestines in consequence of the displacements, severe colicky pains may appear. Prior to this stage, however, the local subjective phenomena consist of distressed sensations of various kinds, attended by the phenomena of neurasthenia.

(*l*) **Fæcal Impaction**.—The pain may be constant or paroxysmal, and is due to irritation excited by the fæcal masses.

(*m*) **Intestinal Obstruction.**—Pains from this cause have already been referred to when describing " constipation."

(*n*) **Intestinal Perforation.**—Here the pain is of sudden onset, and is associated with collapse. It may or may not have been preceded by the clinical phenomena of lesions capable of ending in perforation, as intestinal ulcer and typhoid fever.

The non-intestinal causes of abdominal pain include:

a. Spinal-cord disease.
    i. Tumor of cord or meninges.
    ii. Locomotor ataxia.
    iii. Caries of the vertebræ.
b. Neuralgia of the abdominal walls.
c. Rheumatism of the abdominal walls.
d. Gall-stone colic.
e. Renal colic.
f. Cystitis.
g. Aneurysm of the abdominal aorta.
h. Pelvic disorders in the female.

(*a*) **Spinal-Cord Disease.**—The cases in which the initial symptom of tumor of the spinal cord consists of a bilateral neuralgia of the trunk (chest or abdomen) are rare. Nevertheless they are of sufficient importance to bear in mind in cases of otherwise unexplainable persistent abdominal pain. The abdominal pains of locomotor ataxia constitute one of the crises of this disease, and correspond closely to the gastric crises already described. Abdominal pain from vertebral caries is especially frequent in childhood, and I have seen it give rise to some very absurd diagnoses under the circumstances, *e.g.*, appendicitis, rheumatism, etc.

(*b*) **Neuralgia of the Abdominal Walls.**—This is especially liable to occur in conjunction with or as one of the sequelæ of herpes zoster.

(*c*) **Rheumatism of the Abdominal Walls.**—In this condition the pains are superficial, and consist of a steady soreness, which is aggravated during motion or attempts to move.

(*d*) **Gall-Stone Colic.**—In biliary colic the pain is very severe, and finds its seat of greatest intensity about the region of the gall-bladder.

When the common duct is obstructed, it is attended by jaundice and intermittent fever and the passage of clay-colored stools. Sometimes the paroxysms of pain are attended by vomiting. In some cases the pains radiate to the shoulder and back.

(*e*) **Renal Colic.**—The pain in nephritic colic is due to the passage of a calculus from the kidney through the ureter to the bladder. The pain starts in the region of one kidney and extends downwards to the pubes, and in some cases even into the corresponding testicle and thighs.

(*f*) **Aneurysm of the Abdominal Aorta.**—Pain from this lesion has no definite character, nor is it necessarily present in all cases of abdominal aneurysm. Much depends upon the location of the tumor, and the organs and structures upon which it exerts pressure. The diagnosis is based upon the recognition of the usual physical signs of aneurysm.

(*g*) **Cystitis.**—In cystitis the pain is supra-pubic and at the neck of the bladder. It is associated with ardor urinæ, and relief from urination is but short-lasting. The urine contains pus and mucus.

(*h*) **Pelvic Disorders in the Female.**—All of the utero-ovarian lesions may be associated with more or less severe pain. The diagnosis is made by a physical examination, though special characteristics and situations of the pain may be suggestive *a priori*. While on this subject, it is well to warn the reader concerning the many neurotic manifestations appearing in this locality leading to incorrect diagnoses of organic affections, when neuroses only are present.

(*i*) **Peritonitis.**—The pains of peritonitis are, as a rule, general, and are associated with sensitiveness to touch or manipulation. As peritonitis cannot be a primary disorder, the case must present a history of one of the lesions capable of giving rise to that disease, as appendicitis, gastric ulcer, pyosalpinx.

5. **Tympanites; Meteorism.**—The intestinal tract always contains a certain amount of gas, but this does not accumulate with sufficient rapidity to become a source of discomfort. Nor is it common for it to give rise to any great degree of eructations or escape of flatus per anum. The quantity is not so great but that it can be absorbed by the intestines. The general causes which may produce tympanites are as follows:

a. Abnormal formation of gas in the intestinal tract.
b. Retention of gas within the intestinal tract, formation being normal.
c. Abnormal introduction of gas into the intestinal tract.

The abnormal formation of gas in the intestinal tract is dependent upon fermentation of food-products. Starchy substances, as potatoes, and food rich in cellulose, as beans, carrots, turnips and cabbage, are especially liable to produce intestinal flatulence. The main cause for retention of gas is found in intestinal obstruction from any cause. It may also be due to loss of the muscular tone of the intestinal walls, as in peritonitis and in typhoid conditions generally. Abnormal introduction of gas into the intestinal tract is the cause of the tympanites of hysterical and hypochondriacal individuals. (*Vide* remarks on Flatulence, p. 211.)

6. **The Fæces.**—The clinical study of the fæces embraces observations as to their quantity, consistence, shape, chemical reaction, color, odor, and the presence of non-fæcal matters; *i.e.*, undigested food, foreign bodies, blood, mucus, and fat. Under exceptional circumstances, microscopical and chemical examination of the fæces is necessary.

(*a*) **Quantity of Fæcal Matter Discharged.**—This varies within wide limits, even in perfectly healthy individuals. The average daily quantity in the adult is about five ounces; and yet from two and a half to ten ounces are within the limits of health. The size of the stool depends more on the character and quantity of food taken than on the size of the individual. Thus, vegetable food, particularly potatoes, leaves a large fæcal residue, while animal foods permit of but little waste. It is generally held that about one-seventh of the weight of food taken is excreted by the bowels. In children, however, it will be found that the stools are disproportionately large to the food taken, the reverse condition obtaining in old age.

(*b*) **Consistence.**—The normal adult stool is of firm consistence, and is discharged as cylindrical masses. We speak of it as a "formed" stool. Departures from this standard may occur. Under the influence of prolonged retention within the intestinal canal the fluid portion may be absorbed, and the stool becomes "hard" and "nodular." As a result of increased peristalsis and excessive secretion, the consistence of stools becomes lessened, and, according to the extent of the alteration, we speak of them as "semi-solid," "pultaceous," "loose," or "fluid." "Ribbon-shaped" stools are sometimes found in cases of intestinal stenosis, owing to the fæcal masses being forced through a narrow passage. Such a condition must not be regarded as diagnostic, however, unless it is persistent, for such a shape may result from extreme anal contraction. Then, too, its absence must not be regarded as negativing the possibility of stricture, for the stools may re-form in the rectum after passing the constriction.

(*c*) **Chemical Reaction.**—The normal chemical reaction of the fæces is slightly alkaline or acid, dependent upon the character of food taken and the form of fermentation ensuing. In typhoid fever the reaction is markedly alkaline; in infantile diarrhœa it is usually strongly acid. The clay-colored stools of jaundice are generally of acid reaction, dependent upon the presence of the fatty acids.

(*d*) **Color.**—The color of the stools varies considerably, usually, however, presenting a light- to a dark-brown color. It is modified considerably by the character of the food and the length of time they are retained in the canal. Long retention causes them to become darker. Persons on a milk diet usually have light-colored stools; a greenish tint is presented when the diet is largely vegetable. Green stools are very commonly observed in cases of infantile diarrhœa, owing to acid fermentation. They may be observed exceptionally in cases in which the reaction is alkaline or but faintly acid, in which case the color is believed to be due to a green pigment-forming bacillus. Bright-green stools are also observed in some cases of typhoid fever, especially when the upper portion of the colon is involved.

Certain drugs and foods give the fæces characteristic colors. Thus, iron, bismuth and charcoal cause them to become black; calomel, green; santonine, rhubarb and senna, yellow; hæmatoxylon, red; cocoa in large quantities, gray; spinach, green.

Clay-colored stools are observed whenever the flow of bile is cut off from the alimentary tract, as in cases of obstruction of the gall-duct. Such stools may be associated with a discharge of bile-stained mucus, because there is always associated with it a general cholæmia, and the discharges are stained from the bile-laden blood. Clay-colored stools are occasionally observed in children, as the result of the presence of a large proportion of undigested fat. They are sometimes found in anæmic states and rachitis, because of "the fundamental deficiency of blood-pigment." They are also regarded under appropriate circumstances as suggestive of pancreatic disorder, here being dependent upon "alterations in the mutual reaction of the bile and the pancreatic juice in coloring the intestinal contents."

(*e*) **Odor.**—The characteristic odor of the stools is well known, and is dependent upon skatol and indol. It may be modified by disease. Thus, in cases undergoing acid fermentation, as in infantile diarrhœa, they may present a sour odor; in obstructive jaundice, owing to the absence of bile from the intestinal contents and deprivation of the supposed antiseptic properties of that fluid, the stools become exceedingly malodorous. The odor may be unusually offensive in cases of indigestion. In dysentery and other diseases characterized by ulcerations the stools may assume a gangrenous odor, because of the formation of sloughs. In diseases in which fæcal matters disappear and the discharges consist almost entirely of serum or mucus, odor may be almost absent, as in cholera Asiatica and dysentery. An ammoniacal odor may be present in uræmia, and when there is a fistulous communication between the intestinal and urinary tracts.

(*f*) **Presence of non-fæcal matters,** including foreign bodies, mucus, blood, fat, pus and undigested food, and shreds.

i. **Foreign bodies** are observed only when introduced by accident, excepting in cases of the insane and mentally deficient.

ii. **Mucus** occurs in small quantity under normal conditions. It may appear in large quantities, and is then evidence of a catarrhal condition of the intestinal mucous membrane. The more intimately the mucus is mixed with the fæces, the higher up is the lesion. When the mucus is found coating the fæcal masses, and not mixed with them, it is a fair assumption that the lesion is limited to the sigmoid flexure and the rectum. Its quantity varies greatly in disease. In cholera morbus and cholera Asiatica and dysentery in association with serum it constitutes the main component of the stools.

iii. **Fatty stools** are observed when fat is taken in such excessive quantities that the digestive fluids are unable to take care of it, and in pancreatic disease. In cases of diabetes dependent upon disease of the pancreas, they are also observed. In any morbid state of the intestinal tract in which the epithelial layer is extensively destroyed, fatty stools may result from non-absorption. The appearances presented by fatty stools are various ; thus, they may be simply greasy or clayey, or they may contain solid masses of fat or cylinders of a greasy material which has not inaptly been compared to macaroni.

iv. **Undigested Food.**—The appearance of undigested food in the fæces is known as " lientery." Undigested food in large quantities in the stools can occur only when digestion is seriously at fault or peristalsis exceedingly active. It is not uncommon in healthy persons to find particles of skins of fruits and vegetables, seeds, grains, etc.

v. **Pus** appears in the stools only in exceptional instances, and, according to the associated symptoms, is to be regarded as evidence of ulceration, or of the rupture of an abscess into the intestinal tract. When in any great quantity, the latter is usually the case.

7. **Incontinence of Fæces; Involuntary Defecation.**—The conditions which may give rise to fæcal incontinence include

a. Spinal-cord affections, in which the centre for the sphincter ani is involved.
  i. Locomotor ataxia.
  ii. Ataxia paraplegia.
  iii. Acute and chronic transverse myelitis.
  iv. Disseminated sclerosis.
  v. Spinal meningitis.
  vi. General paralysis of the insane.
  vii. Meningeal and intra-spinal hæmorrhage.
b. Affections in which unconsciousness or defective cerebration is a prominent feature.
  i. Apoplexy.
  ii. The typhoid state.
  iii. Coma from any cause.
  iv. Sunstroke.
  v. Shock.
  vi. Some cases of insanity and imbecility.

- c. Affections characterized by abnormal or perverted muscular action.
    - i. Chorea.
    - ii. Epilepsy.
    - iii. Tetanus.
- d. Affections characterized by active peristalsis and abnormal fluidity of the intestinal contents.
    - i. Acute enteritis.
    - ii. Cholera morbus and Asiatica.
    - iii. Dysentery.
- e. Local conditions of the sphincter ani.
    - i. Lacerated perineum.
    - ii. Fissure of anus.
    - iii. Fistula in ano.
- f. Conditions throwing unusual and unexpected strain on the anal sphincter.
    - i. Violent paroxysmal cough.

8. **Painful Defecation.**—Painful defecation may be the result of the forcing of unusually hard or abnormally large fæcal masses through the anus; or it may be dependent upon local lesions. The latter include fissure of the anus, fistula, hæmorrhoids, rectal ulceration, and proctitis. It may also accompany salpingitis, cancer of the cervix, pelvic peritonitis, coccygodynia, and prostatitis.

Synopsis of the Symptoms and Clinical Course of the Most Important Diseases of the Intestines.

1. Enteritis.

A number of different pathological conditions may be included under the term "enteritis," all of which present inflammation of some one portion or some one tissue of the intestinal structure as their distinctive pathological feature. They are as follows:

- a. Catarrhal enteritis.
    - i. Acute.
    - ii. Chronic.
- b. Phlegmonous enteritis.
- c. Diphtheritic enteritis.
- d. Membranous enteritis.
- e. Ulcerative enteritis.
    - i. Follicular and catarrhal ulcers.
    - ii. Simple ulcerative colitis.
    - iii. Ulceration from external perforation.
    - iv. Cancerous ulceration.
    - v. Stercoral ulcers.

    vi. Solitary ulcer in cæcum or colon.
    vii. Tuberculous ulcer.
    viii. Syphilitic ulcer.
    ix. Dysenteric ulcer.
    x. Amyloid ulcer.
    xi. Embolic and thrombotic ulcer.
    xii. Duodenal ulcer.
f. Enteritis of children.
    i. Acute intestinal indigestion or simple diarrhœa.
    ii. Chronic entero-colitis.
    iii. Cholera infantum.

(*a*) Acute Catarrhal Enteritis.

The attack is preceded by some dietetic error, exposure to cold or wet, the taking of some poison, as spoiled food or irritant poison, or to nervous influences. It may occur as a secondary affection, *e.g.*, following gastric indigestion, portal stasis, chronic heart affections, many of the acute infectious diseases, and cachectic disorders. The symptoms may be of gradual or rapid onset,—usually, however, the latter. Diarrhœa is the first symptom, the stools increasing in frequency and lessening in solidity with the progress of the case. Pain is early associated with the increased intestinal activity. There is always more or less griping, and usually considerable distress and soreness. If the attack is a mild one, the above constitute the only clinical phenomena, and the patient recovers promptly on the removal of the offending materials from the gastro-intestinal tract. In the more severe cases, nausea and more or less prostration appear, and the case continues for several days. Excepting when attacking the aged or infants, acute catarrhal enteritis offers a very favorable prognosis, nearly all cases being well within three or four days.

Chronic Catarrhal Enteritis presents about the same clinical phenomena as the acute variety, excepting that the constitutional symptoms, by reason of the long continuance of the affection, are more profound. It usually results from the too frequent repetition of acute catarrhal enteritis, or from a long continuance of the causes of that affection. In some instances the disease is characterized by alternation of diarrhœa and constipation. The stools of chronic enteritis are more likely to contain blood, pus and mucus than those of the acute form. The mucus only is characteristic.

(*b*) Phlegmonous Enteritis.

In this disease we have a condition which cannot be diagnosed during life, excepting as it occurs in conjunction with intussusception and strangulated herniæ, which are its most common causes. It is especially liable to attack the duodenum, and leads to suppuration in the submucous layer of the intestines and abscess formation. In a general way the symptoms are those of peritonitis.

(*c*) Diphtheritic Enteritis.

This is always a secondary process, occurring either in the course of some of the acute infectious diseases—*e.g.*, pneumonic fever, pyæmia, or typhoid fever; as a terminal process in chronic diseases, as Bright's disease, cirrhosis of the liver, and cancer; or as an effect of certain poisons, as lead, mercury and arsenic. The symptoms are not at all diagnostic; indeed, it may run its course without symptoms, the disease being discovered unexpectedly at the autopsy. Unless the stools contain shreds of membrane, no diagnosis can be made.

(*d*) Membranous Enteritis.

This, in the majority of cases, is a membranous colitis, although observations demonstrate that the intestines as high as the duodenum may be involved. The literature relating to this affection is in a sadly confused state, and it hardly seems possible that authorities can be describing the same affection under this title. Of all descriptions, that of Goodno is unquestionably the clearest. At any rate, that author describes one clinical entity worthy of the name "membranous enteritis." It occurs almost exclusively in neurotic women past the age of forty. It is characterized by paroxysms of abdominal pain, tenesmus, digestive and nervous disturbances, with the discharge of pseudo-membranous formations by the bowels. There are free intervals in which the intestinal symptoms are absent, and the patient exhibits only the manifestations of hysteria or neurasthenia. Usually general nurition is seriously disturbed. The disease is not apt to terminate fatally, but it runs a prolonged course, and is exceedingly resistant to treatment.

(*e*) Ulcerative Enteritis.

This includes a variety of intestinal conditions having ulcerations as their common characteristic. They include (i) follicular ulceration; (ii) simple ulcerative colitis; (iii) ulceration from external perforation; (iv) cancerous ulcers; (v) stercoral ulcers; (vi) solitary ulcer in the cæcum or colon; (vii) tuberculous ulcer; (viii) syphilitic ulcer; (ix) dysenteric ulcer; (x) amyloid ulcer; (xi) embolic and thrombotic ulcer. The differentiation of these, in practice, is always a difficult if not a nearly impossible undertaking. The special clinical features of ulceration are identical in all cases. We may sometimes reach a correct conclusion as to the origin of the ulcerative process by reason of etiological factors and associated clinical conditions. The *catarrhal* and *follicular* ulcers are the most common, and are situated for the most part in the large intestine. They occur in conjunction with intestinal catarrhs. The *follicular* ulcers, starting in the follicles, not infrequently progress to suppuration and rupture. Both the catarrhal and follicular ulcers may coalesce to form large ones. The *stercoral* or *decubital* ulcer is located in the large intestines, its favorite sites being at points at which fæcal pressure is greatest, as in the bends of the colon, the cæcum and the sigmoid flexure, and stenosed portions of the intestines. The *tubercular* ulcers may, though rarely, occur as a primary intestinal tuberculosis; usually, however, it is secondary to tuberculosis elsewhere. They are especially liable to invade the lower part of the ileum and the cæcum. Nevertheless, they may invade any portion of the intestinal tube. They exhibit but slight tendency to heal. The chronic *dysenteric* ulcer invades the large intestine, and is multiple. They are deep, with raised and undermined edges. They are associated with a high grade of catarrh of the large intestines. They are always preceded by acute dysentery. *Syphilitic* ulcers of the intestines constitute one of the rare phenomena of visceral syphilis. They are located in the majority of cases in the large intestines, and are due to the breaking down of gummata of the intestinal walls. They tend to spread in surface rather than in depth. *Embolic* and *thrombotic* ulcers originate in the occlusion of one of the small branches of the mesenteric arteries. Localized necrosis, varying in extent according to the surface deprived of its supply, ensues. They are especially liable to occur as the result of septic endocarditis.

It is hard to construct a well-defined clinical picture of intestinal ulceration, as cases vary greatly in their symptomatology. Many times there is entire absence of subjective discomfort, and the existence of the trouble is not even suspected. Usually, however, pain is present. Tenderness is even more common, especially in cases of tuberculous ulceration. In chronic dysenteric ulceration, the tenderness is most marked over the descending colon and sigmoid flexure; in tuberculous ulcer, at or about the umbilicus. In any case, the spontaneous pain and the tenderness on pressure is greater as the ulceration reaches nearer to the peritoneal surface. The objective symptoms relate entirely to the appearance of the stools, and consist of the appearance of pus, blood, and portions of the intestinal mucous membrane, in the fæces. Accidental but diagnostic symptoms are tympanites, fever, and general malnutrition.

(*f*) Enteritis of Children.

i. **Acute Intestinal Indigestion or Simple Diarrhœa.**—The acute dyspeptic diarrhœa of infancy and childhood is brought about by excessive or improper food, especially in hot weather. Constitutional factors, as a tubercular or syphilitic inheritance or weak digestive organs, act as predisposing causes. The symptoms consist of diarrhœa, the stools appearing with varying frequency, and ofttimes associated with vomiting, pain and flatulence. In mild cases the patient recovers promptly after complete evacuation of the digestive tract. Fever may be present. Usually it is moderate in degree, and, even when high in the beginning, it is not of long continuance. The general nutrition of the child is not greatly disturbed. It is to be differentiated from the other diarrhœal affections of infancy by the absence of mucus and serum in the stools.

ii. **Chronic Entero-Colitis; Chronic Diarrhœa of Infancy.**—Predisposition in the shape of a tubercular or syphilitic inheritance or the rachitic constitution plays a more important part in the etiology of chronic entero-colitis than in simple dyspeptic diarrhœa. The exciting causes include persistent neglect of proper methods of feeding, bad ventilation, and defective hygiene. Chronic entero-colitis is of gradual onset. The symptoms include diarrhœa, flatulence, and general impoverishment of nutrition. The stools contain particles of undigested food, and are of watery consistence. Mucus, and even blood, are found in the stools after the disease has continued for a few days. Gastric digestion may be disturbed. It is usual, however, to find the patient's appetite undisturbed, leading not infrequently to overloading of the stomach. The latter is unfortunate, as it serves only to add to the intensity of the pathological process in the intestines. The general malnutrition is exhibited by wasting, sunken features, listlessness, constant moaning, and thrush.

iii. **Cholera Infantum.**—This is an acute milk infection, for it can only occur in infants who have partaken of milk contaminated with bacteria. It is essentially a disease of the hot season. In rare cases only does it occur in breast-fed babies; but, even in them, investigation will show that the food-supply has been infected. The onset of cholera infantum is always sudden, and is characterized by profuse vomiting and purging,—so severe, indeed, as to lead to constitutional depression in an incredibly short space of time. At first the vomited matters consist solely of food; but, as the stomach is emptied of these, they are composed of mucus and bile. The stools are at first fæcal in character, but with the progress of the disease assume a more and more watery character, until finally they consist only of serum. Fever is a prominent feature, the thermometer registering 103° to 105°. The disease is a very dangerous one. In fatal cases the patient becomes unconscious and develops a condition of extreme exhaustion, with irregular pulse and respiration, and dies.

iv. **Acute Entero-Colitis.**—This disease, likewise, is due to milk infection,—the poison, however, probably being different from that occasioning cholera infantum. The symptoms may be either rapid or gradual in their onset, usually the latter. Diarrhœa is the first manifestation of ill-health. The stools become more frequent, and are soon found to contain mucus in large quantities. If the case is a severe one, streaks of blood will also be found in the stools. Vomiting, pain and flatulence are associated symptoms. The general condition of the little patient is much disturbed, emaciation being rapid and extreme. Fever is commonly present, but does not assume the high grade observed in cholera infantum.

(*g*) Cholera Morbus.

This is a hot-weather disease. It is especially liable to occur in seasons during which hot days are followed by cool nights. In many instances it seems to be due to an intoxication arising from the taking of food which has undergone bacterial decomposition, or indulgence in unripe food, ice-water when overheated, etc. The symptoms are always of sudden onset, and consist of violent vomiting and purging. At first the ejected matters consist of the normal contents of the digestive tract. Soon the matters vomited consist only of mucus and bile, and finally of large quantities of watery substance. The stools become more and more liquid, until finally they lose their fæcal character entirely, and consist solely of mucus and serum. Pain is a prominent feature throughout the course of the disease. It usually precedes the vomiting and purging. Not infrequently painful cramps of the calves and other portions of the body are experienced. Constitutional disturbance is in evidence, as shown by the great prostration, the general pallor, and the cold sweat. This disease closely simulates cholera Asiatica,—from which, indeed, it can only be differentiated by the knowledge of the presence or absence of an epidemic of that disease.

2. Appendicitis.

Etiological factors do not play any part in the diagnosis of appendicitis. This arises in great part from our almost entire ignorance of the causes which give rise to this disease. We know, of course, that the majority of cases occur in young subjects ; but other ages are sufficiently liable to cause us to be on the lookout for appendicitis at any time of life from infancy to old age. Males are more predisposed than females in the proportion of four to one, because, as explained by Van Lennep, the appendix in the female derives an additional blood-supply through the appendiculo-ovarian ligament. Among the exciting causes are accumulation of fæcal matters in the appendix, indiscretions in diet in those predisposed by reason of previous attacks, constipation, alterations in its position interfering with its blood-supply, and infection,—mainly by the bacterium coli commune and the streptococcus pyogenes. There is scarcely a disease capable of producing such a variety of clinical pictures. The character of the pathological changes, of course, have much to do with this ; and yet one cannot say with certainty, prior to operation, whether a case is catarrhal or ulcerative. In the majority of cases appendicitis is of sudden or rapid onset, the first symptoms consisting of abdominal pains, ofttimes colicky in character, with sensitiveness at the McBurney point, and sometimes nausea, vomiting, and constipation. Fever, though present, is not a reliable guide as to the severity of the attack. The pain, though constant, is subject to paroxysmal exacerbations. Quite naturally, too, we find rigidity of the abdominal muscles, especially of those on the right side. One type of cases, generally known as *appendicular colic*, is attended by all the symptoms above enumerated with the exception of the fever. In the majority of cases the pulse is quickened, and presents other features marking the serious nature of the illness. But too much reliance must not be placed upon this, for cases are observed in which the pulse shows scarcely any alterations. The tongue is usually furred, and moist and flabby.

In addition to the sensitiveness at McBurney's point, some cases may exhibit a boggy, ill-defined mass in the right iliac fossa; sometimes it is possible to feel the swollen appendix when the abdominal walls are thin, but such cases are exceptional. The disease may progress to recovery, to the development of suppuration, or to a general peritonitis. In many instances the attack does not clear up entirely, but leaves the patient with a sense of ill-defined discomfort in the right iliac region or sensitiveness over McBurney's point; or recovery may be complete and the patient never have another attack; or there may be recurrences from time to time, each one being more severe than its predecessors. It seems scarcely possible to mistake appendicitis for any other disease; indeed, it would be impossible, if attention is paid to the centralization of the pain about the McBurney point.

3. Intestinal Tumors.

With very few exceptions, tumors of the intestines are malignant. The vast majority of cases attack the large intestines; and, of these, nearly all are of the rectum. In a general way it may be said that their symptoms are the constitutional manifestations of malignant disease in other portions of the body, to which may be added the phenomena resulting from disturbed intestinal function, *i.e.*, the symptoms of intestinal obstruction. Other than this, it is impossible to construct a clinical picture of intestinal tumors, because the symptoms vary greatly, according to the portion of the tube involved. For purposes of description, we may divide cases of cancer according to their location as follows:

- i. Cancer of the duodenum.
    - 1. Suprapapillary cancer.
    - 2. Infrapapillary cancer.
    - 3. Circumpapillary cancer.
- ii. Cancer of the jejunum and ileum.
- iii. Cancer of the large intestine, exclusive of the rectum.
- iv. Cancer of the rectum.

i. **Cancer of the Duodenum.**—1. *Suprapapillary Cancer.*—By reason of the resulting occlusion of the duodenum there is interference with the ability of the stomach to empty itself. Necessarily, then, we have all the symptoms of impaired gastric motility. Physical examination may discover a tumor which is fixed, and is located in the right hypochondrium. After this tumor has existed for a length of time, dilatation of the duodenum and stomach ensues. Unlike cancer of the pylorus, there is little or no lessening of the hydrochloric acid percentage in the gastric contents, and lactic acid is absent from the same. Still, too much importance must not be attached to the gastric analysis, because the dilatation of the stomach may result in such alterations of the gastric secretion as to bring about the changes characteristic of gastric cancer.

2. *Infrapapillary Cancer.*—The one characteristic symptom of infrapapillary duodenal cancer is persistent and long-continued vomiting of bile. The reason for this is evident, as the intestinal stenosis is immediately below the point at which the gall-duct empties. The tumor, when palpable, is immovable. Examination of the gastric contents will show the permanent presence of bile therein. Quite naturally, there is dilatation of the duodenum and stomach.

3. *Circumpapillary Cancer.*—The subjective symptoms when the tumor is circumpapillary are not of much diagnostic importance. They include pain and a variety of manifestations indicating disordered digestive functions. The most obtrusive symptom is the jaundice, which develops suddenly and without pain, though occasionally there may be pain strongly suggestive of biliary colic. When the jaundice once appears, it is persistent and progressive. The tumor is usually so deeply situated as not to be discoverable by palpation. The liver is not enlarged, and the gall-bladder is decreased in size.

ii. **Cancer of the Jejunum and Ileum.**—The diagnosis of cancer in this situation is not possible until symptoms of intestinal obstruction appear. The tumor is not palpable until the lesion is well advanced, and is very movable.

iii. **Cancer of the Large Intestine, Exclusive of the Rectum.**—Pain is practically always present, and may be distinguished in each case as of two distinct varieties, namely, one which is constant and is due to the tumor *per se*, and the other paroxysmal or colicky. The tumor pain is usually dull and fixed; occasionally, however, it may radiate in various directions. The paroxysmal pains vary greatly in all their characteristics, even in the same case. They generally centre around the location of the tumor, and tend to grow more and more severe with the progress of the case, until finally they are unbearable, and recur at such short intervals as to be almost practically continuous. Constipation is usually present and aggravates the pain. Vomiting becomes a symptom as soon as there is intestinal stenosis, and is in direct proportion to the extent of that condition. Sometimes, in the early history of the case, the condition of the bowels is one of constipation alternating with diarrhœa.

Speaking of the value of the above-mentioned symptom as diagnostic of carcinoma of the large intestine, Boas says: "There are three cardinal symptoms, viz.: obstinate constipation or constipation followed by diarrhœa, colicky paroxysms, and vomiting. Increasing experience has shown me that, even without the presence of a palpable tumor, these three symptoms frequently denote intestinal cancer. The frequency of the attacks, the characteristic increase of the paroxysmal pain, increasing constipation, the presence of unmistakable cachexia even in this stage, and marked loss of body-weight, all point toward the correct diagnosis." Nevertheless, one can hardly feel satisfied unless a tumor is palpable. This is said to be possible in about forty per cent. of the cases. The tumor is hard, nodular, tender on pressure, and movable. When palpation fails to discover the presence of a tumor in a suspected case, the examination should be repeated on a subsequent occasion after thorough evacuation of the bowels.

Diagnostic errors in cases of intestinal cancer arise not only from the variety of the symptoms, but from misconceptions as to certain of them. Thus, the presence of blood in the stool while of importance to a certain extent, is present in less than one-fourth of all cases. Then, too, while we expect constipation as the usual condition of the bowels, diarrhœa may exist, indeed is not uncommon, when the growth has begun to ulcerate, especially if it is situated high up in the colon. The most reliable symptom—the one that makes the diagnosis absolutely certain—is the passage of portions of the growth per anum.

Certain atypical cases may be said to be unrecognizable during life. As in internal cancers in other portions of the body, the disease may run its course from start to finish without pain or other symptom or sign to indicate the seat of disease, but with the emaciation and cachexia so characteristic of malignant disease. In still other cases, no symptoms are observed until there is a sudden advent of intestinal obstruction.

4. Dilatation of the Colon.

Hale White divides cases of dilatation of the colon into four classes, as follows:

i. *Temporary dilatation from flatulent distention.* This variety may give rise to considerable inconvenience from pressure upon adjacent organs, but it disappears promptly under treatment.

ii. *Distention of the colon by fæcal matter.*

iii. *Dilatation of the colon from obstruction* below the dilated portion. In these cases the size of the colon may reach enormous dimensions. The usual cause of the obstruction is a tumor.

iv. *Idiopathic dilatation of the colon*, the origin of which is obscure. Some cases have been demonstrated to be due to narrowing of the intestines low down.

5. Intestinal Neuroses.

i. **Motor Neuroses.**—The motor neuroses of the intestines may be tabulated as follows:

1. Misdirected motility.
    - Enterospasm.
    - Peristaltic restlessness.
2. Defective motility.
    - Paresis or atony of the intestines.
    - Neurotic flatulence.
3. Nervous diarrhœa.
4. Intestinal neurasthenia.

Enterospasm, Including Proctospasm.—Enterospasm is a very painful condition. It is manifested clinically by paroxysms of painful intestinal contractions, associated with rumbling and borborygmi. There is strong urging to stool, but only small scybalæ are passed. It is liable to be confounded with organic stenosis, from which it may be distinguished by its long duration, the presence of a highly neurotic constitution, the good physical condition of the patient, and the absence of a history of a possibility of a stenosing lesion.

In *proctospasm* the pain is limited to the rectum, and is associated with strong contraction of the sphincter ani.

Peristaltic Restlessness; Tormina; Intestinorum Nervosa.—This condition is decidedly more common than the preceding one. It is exhibited by exaggerations of the intestinal peristaltic movements, even to the extent of producing pain. These movements are visible by the physician. When not so, they may be readily excited by abdominal friction, the pouring of ether upon the abdomen, or the distention of the intestines by air or carbonic acid gas. The duration of the paroxysms is quite variable. They are not infrequent at night, keeping the patient awake.

Paresis or Atony of the Intestines; Atonic Constipation.—Here we have what is probably the most frequently observed intestinal neurosis among women. It is usually associated with a general constitutional sluggishness of function. Many of these patients suffer also from manifestations of hysteria, hypochondriasis and melancholia. In some instances these phenomena are the result of the constipation; in the majority it is my belief that all the symptoms are the result of the cause giving rise to the intestinal atony. Sometimes the intestinal atony is produced by long-continued abuse of enemata. Boas suggests, as the best method of demonstrating the presence of atony, the injection of a measured quantity of water into the bowel, and eliciting splashing sounds in the corresponding segments of the large intestines.

Nervous Flatulence.—In this we have an exceedingly resistant intestinal neurosis of obscure origin. The associated symptoms are those resulting from the accumulation of gas. There is abdominal discomfort and distention. Relief from expulsion of gas is but partial; psychic and hysterical manifestations are commonly present.

Enteralgia.—This shows itself by paroxysms of pain of variable duration, without local objective conditions to account for the same. Constipation is generally the prevailing condition of the bowels. The pain is usually so severe as to force the patient to take to his bed. The usual causes are found in locomotor ataxia (intestinal crises), lead-poisoning, arthritism, hysteria, neurasthenia, and malarial poisoning.

Nervous Diarrhœa.—This is commonly associated with more or less nervous debility. The best known example of this affection is the diarrhœa prevalent among medical students at or about the time of their examinations. The associated symptoms are practically the same as those accompanying catarrhal and other diarrhœas. In most instances the attacks are explosive in their onset, and bear evident relationship to mental influences.

Intestinal neurasthenia presents a wide range of symptoms, the principal of which include constipation, abdominal distention, and expulsion of large quantities of odorless and tasteless gas by way of both mouth and anus. With these as the central phenomena are the well-known stigmata of neurasthenia and hysteria.

6. Intestinal Obstruction

Has already been described when speaking of the causes of constipation. (*Vide* page 245.)

7. Duodenal Ulcer.

The special symptoms of duodenal ulcer include pain, tenderness, vomiting, and intestinal hæmorrhage or hæmatemesis. The pain comes on several hours after the taking of food, and, as has been pointed out elsewhere (page 253), is relieved by eating. The tenderness to palpation corresponds to the seat of the pain. The associated vomiting is reflex, and is the result of the severity of the pain. Hæmorrhage is a frequent symptom, probably occurring in about half the cases. The symptoms, it will thus be seen, are strikingly similar to those of gastric ulcer; but the relation in point of time between the pain and the taking of food is different. The differentiation of duodenal ulcer and hyperchlorhydria is not so easy. In fact it would seem, in the absence of hæmorrhage, that time alone will enable one to reach correct conclusions. The suggestion that the failure of properly-directed treatment for hyperchlorhydria should lead to the adoption of therapeutic measures against duodenal ulcer is a good one.

8. Acute Infectious Diseases, the Prominent Symptoms of which Relate to the Gastro-Intestinal Tract.

Two diseases may be included in this group, as follows:

(a) Dysentery.
(b) Cholera Asiatica.

(*a*) Dysentery.

Dysentery is to be regarded for the present at least as a generic term, descriptive of the clinical phenomena attendant upon no less than three distinct pathological conditions, as follows: (i) Catarrhal dysentery; (ii) amœbic or tropical dysentery; and (iii) diphtheritic dysentery. Any one of these three varieties may become chronic by reason of persistent inflammatory changes in the intestines leading to their infiltration and ulceration.

The ordinary catarrhal cases result from sudden changes in temperature, such as are experienced in autumn, when hot days are succeeded by cool nights, exposure of various kinds, nervous influences, dietetic irregularities, and obstinate constipation. The amœbic variety cannot be produced excepting by infection. It is a disease of the tropics, not being observed farther north than 40° latitude.

The symptoms of dysentery include fever, frequent evacuations of the bowels, the stools consisting finally of mucus and blood, and attended by marked tenesmus recti and great prostration.

The fever of dysentery is not usually of high grade, nor is it often absent. Usually it is ushered in by a slight rigor, followed by a rise of temperature of two or three degrees. When continued for more than two or three days the presence of some complication is to be suspected. When associated with malaria, which is by no means uncommon, the temperature may reach 104° F. or more. When the dysentery is of the gangrenous type, then the fever will partake of the character belonging to that pathological process. The incidence of hepatic abscess brings with it the characteristic temperature-curve of suppuration. Fever can hardly be regarded as of much diagnostic or prognostic value in the clinical study of uncomplicated cases of this disease.

The disturbance of intestinal function is noticed from the first day of the disease. In the beginning, the stools are like those of an ordinary diarrhœa; but they soon lose their fæcal character, and finally consist of nothing but mucus and blood. At this time they are odorless. The severe rectal tenesmus forces the patient to stool at very short intervals, but the quantity evacuated each time amounts to hardly more than a few drachms. In the diphtheritic cases the stools usually present a gangrenous odor, because of the presence of sloughs.

The rectal tenesmus is a source of great suffering to the patient, owing to its constancy. In severe cases it is not relieved by an evacuation. Not infrequently it produces secondary changes about the rectum and anus, as inflammation and fissures. Sometimes the tenesmus extends to the bladder, and then the patient's sufferings are augmented by the ardor urinæ.

The diagnosis of dysentery should never be a difficult matter, as the symptoms are very well defined. The only diagnostic errors are those resulting from the foolish habit some practitioners have of calling every case of diarrhœa attended by straining at stool dysentery.

The catarrhal variety offers a favorable prognosis, and is not likely to be followed by sequelæ. The amœbic variety has exhibited as sequelæ or complications abscess of the liver, circumscribed and profuse peritonitis, intestinal strictures, pyæmia, septicæmia, and arthritis. The diphtheritic variety is almost invariably fatal.

(*b*) **Cholera Asiatica.**

The etiology of cholera Asiatica may be summed up in a few words—infection by the comma bacillus of Koch. This infection is carried almost entirely by drinking-water. In other words, cholera is a water-borne disease. Some cases are undoubtedly carried by house-flies. Epidemics seem to be favored by high temperature, as the disease occurs with especial frequency in lowlands and in hot countries.

The clinical course of cholera Asiatica may be studied as consisting of three stages, namely: (i) Preliminary diarrhœa; (ii) stage of collapse; (iii) stage of reaction. The *preliminary diarrhœa* presents no characteristic features beyond the severity of the constitutional disturbance. There are frequent loose stools, with colicky abdominal pains and vomiting. Some few cases terminate favorably with this period. They are commonly known as *cholerine*. The *stage of collapse* follows immediately upon the diarrhœa, and without any warning; the stools become watery, and are repeated at very short intervals. They may or may not be associated with pain. Very shortly the patient becomes exhausted to the verge of collapse. Vomiting sets in; and, like the stools, the vomited matters are of watery character. This serves to still further add to the exhaustion. The patient is now collapsed, with sunken features and cold and shrivelled surface. The pulse becomes weak and rapid, ofttimes imperceptible. Notwithstanding the external evidence of subnormal temperature, it is usually the rule to find the rectal temperature to be above normal. Many of the symptoms of this stage of cholera are the direct result of the dehydration of the body by reason of the profuse watery discharges. The mouth becomes dry; urine is suppressed; indeed, all secretions are temporarily abolished excepting the sweat. The usual duration of this stage is from twelve to twenty-four hours. The disease may, however, terminate fatally within three or four hours, or even before the watery diarrhœa or rice-water discharges appear. The *stage of reaction* is characterized by the gradual lengthening of the intervals between the bowel movements and the cessation of vomiting; the surface coldness disappears; the various secretions are restored; and usually the patient is on the road to health. Even at this stage of the disease recovery is not assured, for a severe diarrhœa may set in and end fatally. Another condition sometimes appearing at this time is known as *cholera typhoid*—the patient being delirious, with dry tongue and rapid and feeble pulse, death supervening in coma.

The only affection with which cholera Asiatica may be confounded is cholera morbus or cholera nostras. The symptoms of the two diseases are identical. A differentiation is only possible by means of a bacteriological examination of the stools, which in the case of cholera Asiatica will contain the comma bacillus.

Cholera Asiatica is always a dangerous disease, the mortality ranging in different epidemics from 30 to 80 per cent.

The Rectum.*

The carelessness manifested in the examination of patients suffering from rectal diseases seems to be based upon a lack of knowledge of the great variety of conditions which may produce symptoms and lesions in this locality. It is too often assumed that the pathology of the rectum begins and ends with hæmorrhoids; hence any local discomfort is diagnosed off-hand as due to that affection. Sometimes the existence of fissure and fistula is borne in mind. Careful attention to examination technique, while demonstrating that these are the most frequently encountered lesions of the rectum and anus, will also discover the possibility of numerous other conditions.

It is of the greatest importance that the rules governing examinations in general be rigidly adhered to in the case of rectal diseases. The patient should be permitted to tell his own story without interruption, after which the physician may question him in order to obtain further details. In these additional inquiries, special attention should be paid to the elucidation of the following points:

1. If pain is present, its character must be determined; its duration noted; and its relation to the act of defecation—whether before or after the act—established with certainty.
2. The condition of the bowels, as to regularity of movement, together with the consistence, color and form of the stools.
3. Conditions associated with defecation, especially as related to protrusions from the anus, these latter including mucous membrane, morbid

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* For the data in this section the author acknowledges his indebtedness to his friend, Dr. T. Louis Adams, of Philadelphia.

growths, and hæmorrhoidal tumors. When such protrusions are present, it must be determined whether or not they become spontaneously reduced, or whether they remain strangulated or semi-strangulated.

4. The character of discharges other than fæcal. These may be bloody, mucous, sanguino-purulent, muco-purulent, or purulent.

5. The power of the sphincter ani, whether competent or incompetent.

6. The association of the rectal condition with disease of the gastro-intestinal tract or liver.

7. The presence or absence of a syphilitic or tubercular history.

The above facts having been determined, it is next in order to proceed with the physical examination. Position of the patient is of the highest importance, for unless this is correct, the observations will certainly be deficient. In the majority of cases, the Sims position will be all that is required; but when it is desired to explore the deeper portions of the rectum, the knee-chest position is essential.

The light should always be daylight by a window facing north. Sunlight, by casting shadows, invalidates the observations. Electric light within a speculum is not to be thought of for the same reason.

Proceeding now with the examination, attention should be directed to the following points:

1. Note the presence of tenderness, redness and swelling. If manipulation produces pain, determine whether it is deep or superficial.

2. The presence of fistulous outlets.

3. The presence of protruding hæmorrhoids or prolapsed mucous membrane.

4. The presence of thrombi, anal folds, or fissures, which may be plainly visible or brought into view by gentle traction upon the anus with the accompanying sentinel pile.

5. The character of the discharges, whether bland or excoriating, and their effect upon the anus and surrounding tissues.

6. Glandular involvements (femoral and inguinal glands) as evidencing malignant or syphilitic conditions, and general systemic disturbances.

Digital and Instrumental Examination.—This demands attention to the following points:

1. Before proceeding with the digital examination, the anus should be thoroughly cleansed with carbolated green soap or synal; after which it should be rinsed with a bichloride solution.

2. Inject into the rectum one-half drachm of a 2 per cent. solution of cocaine, and wait five minutes in order to secure its anæsthetic effects.

3. Lubricate the finger thoroughly with carbolized cosmoline or any other antiseptic lubricant.

4. Note the condition of the sphincter; its power of resistance, whether

normal or intensified; note its size, whether hypertrophied or not. Hypertrophy of the sphincter is to be recognized by the increased resistance of the muscle, and by its depth, which may be determined by taking it between the index finger in the anus and the thumb on the cutaneous surface.

5. After the finger is well introduced, note the condition of the mucous membrane. If in a normal state, it should be smooth, distensible, and elastic.

6. No thought should be had for the presence of internal hæmorrhoids, for *their presence can be determined by inspection only.*

7. Note the calibre of the rectum and the presence of any new growths.

8. Note the condition of the prostate gland.

9. The educated finger will also readily detect the presence not only of deep-seated abscesses, but also the opening of internal fistulæ. The latter usually present tumefied edges. The calibre of the fistula may vary to such an extent that in one case it may be able to take a fine probe, or it may be very irregular and sufficiently large to admit the finger. The tissues surrounding the opening are hardened and inelastic, although this remark does not apply with equal force to the internal opening.

10. For examination of the upper third of the rectum, the middle finger may be used, with the patient in the standing position and directed to strain down.

11. The use of the sound is also important for detecting conditions existing in the upper third of the rectum and at its junction with the sigmoid. It should be used, however, with great caution, as, if extensive ulceration exists and the rectal walls have become friable, there is a good chance of perforation if undue force is used.

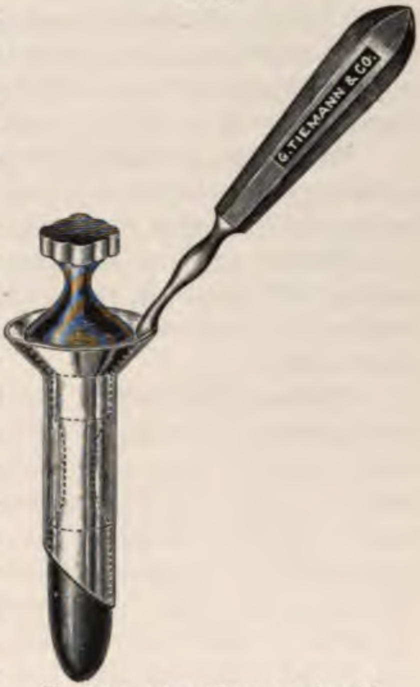
Instrumental Examination.—This involves the use of the speculum, of which various kinds have been suggested, including the tubular, the bivalve, the modified Sims, and the fenestrated.

The one most satisfactory for all purposes is the cylindrical, modelled after the Ferguson vaginal speculum, but provided with a handle set at such an angle as to permit of clearing the buttock while exploring the rectum. It should be provided with an obturator to facilitate introduction. The operator should possess himself of various sizes, both in length and calibre, to suit different conditions of normal and diseased rectums.

As 80 per cent. of all rectal diseases lie within the region of "the pile-bearing inch," it will be seen that such an instrument will be sufficient for almost all purposes of examination and treatment. Its advantages over other specula are that each and every portion of the rectal mucosa can be examined with the one introduction, for the reason that it can, without giving distress to the patient, be rotated, thus bringing into view the various normal and diseased conditions. This is not true of any of the other instruments, as a withdrawal and re-introduction are necessary by reason of the fact that the mucous membrane or any growth that may exist is liable during the rotation to be caught in the fenestra.

The speculum, after having been rendered thoroughly antiseptic and well lubricated, should be introduced to its full extent, *i.e.*, above the pile-bearing inch; after which the obturator should be withdrawn, and, with cotton applicator, the exposed rectal surface be thoroughly cleansed. Then by a slow rotary motion, at the same time gradually withdrawing the instrument, note all conditions presenting, as each will prolapse into or present itself at the end; the lesions which may thus be recognized including hæmorrhoids, ulcerations, constrictions, growths, as benign, malignant, and polypi, fistulous openings, pockets, and foreign bodies.

FIG. 45.



The Adams sigmoidoscope and rectal speculum.

Studying briefly some of the symptoms to which reference has been made above,—space will not permit an elaborate study of the subject,—we come first to speak of **rectal pain.** This symptom is too commonly accepted as evidence of hæmorrhoids, and a diagnosis is not infrequently made accordingly without taking the trouble to make a physical examination. As a matter of fact, all lesions in or below the "pile-bearing inch" produce pain, generally during or after defecation. Usually the pain is experienced most severely after defecation. The explanation for the painfulness of lesions in this locality is found in the free distribution of sensory nerves derived from the sacral and lumbar plexuses.

Practically no pain is present in lesions above the pile-bearing inch in the majority of cases.

When there is extensive ulceration involving the region above and the upper portion of the pile-bearing inch, defecation, by reason of emptying the rectum of the fæcal accumulation and removing a source of irritation, will give great relief.

The clinical value of symptoms relating to the appearance of the fæces has been studied in previous paragraphs. (*Vide* page 256.)

Protrusions from the anus, as already stated, include hæmorrhoids, prolapsus, and morbid growths. Of these, hæmorrhoids are by all odds the most frequently observed.

As to **discharges,** a flow of pure blood is a common attendant upon hæmorrhoids; but it also may be observed in cases of fissure, malignant disease, and the various ulcerations. In the latter two, the blood is in most instances mixed more or less with mucus and pus. The bleeding in hæmorrhoids usually takes place after stool, and ceases in a short time. But it may be very persistent and large in quantity. It may be either arterial or venous. The former is characterized by the continued spurting from a broken down hæmorrhoidal artery, while the latter is an oozing which follows a complete or semi-strangulation. Still, venous oozing may be present without protrusion in the earlier stages of internal hæmorrhoids. It is possible for spontaneous hæmorrhage to occur in conjunction with hæmorrhoids, but such a condition is sufficiently rare to be a clinical curiosity.

Malignant growths produce a more or less constant oozing. The quantity of blood lost is usually not large. There is commonly a mixture of mucus and pus, especially as the lesion progresses towards the fatal issue.

Fissure produces a discharge of pure blood, but only during and shortly after stool. In the earlier stages, the bleeding is apt to be more profuse than later, at which time it may be entirely absent or strictly confined to stool.

Purulent discharges may be observed in all lesions characterized by ulceration and abscess formation. **Abscesses** involve one of three locations, namely: ischio-rectal, marginal, and pelvic. All of these may be latent, *i.e.*, they may occur without giving rise at first to subjective symptoms or functional disturbance to indicate to the patient that he is not in good health; the diagnosis is then possible only by a physical examination. They must necessarily, unless properly evacuated, terminate in fistula. If permitted to rupture spontaneously, a fistula is inevitable. The predisposing causes include thrombi and rupture of blood-vessels, but infection is the determining cause.

The **sanguino-purulent discharge** is observed in the later stages of malignant disease. It is attended by a peculiar odor, which is readily recognized by the experienced, and may be compared to new-mown hay. Later, when we have necrosis of tissue, it becomes very offensive. This discharge is frequently spontaneous, and occurs independently of stool.

A **mucous discharge** is evidence of proctitis. The latter, however, is never a primary condition, being due to some previous lesions, as hæmorrhoids.

Polypi likewise produce a mucous discharge, but these tumors are always evidence of an advanced stage of mucous membrane changes. The soft "tooth-shaped" polypi occur at the junction of the cutaneous and mucous surface. The large villous polypi may occur anywhere.

Ulcerations of the rectum include the syphilitic, the tubercular and the traumatic. Syphilitic ulcerations may be primary, secondary or tertiary. Occurring above the pile-bearing inch, they almost invariably result in stricture. As a rule, they are not painful. Pain, when present, is usually the result of forcing fæcal matter through the attendant narrowing. Tubercular ulcerations are not common, and as a rule are painless.

The **tumors** of the rectum include malignant disease and polypi. The former is readily recognized by the attendant induration, and the glandular involvement which is usually manifested in the groins. Constitutional disturbance is, as a rule, not an early symptom.

Strictures may be traumatic, malignant, or syphilitic. Many of the traumatic cases are the result of heroic measures adopted in the removal of piles by clamp and cautery or the Whitehead and Pratt operations, and in the opening up of fistulous tracts and multiple division of the sphincter. The diagnosis of stricture is made entirely by physical examination.

Ballooning of the rectum is recognized by the non-elasticity of the rectal wall, and the increased calibre of the cavity, which can be recognized by the sweep of the index finger. It is an important symptom of stricture, which occurs above the ballooning. In intestinal obstruction, it may occur below the stricture.

CHAPTER VI.

THE LIVER.

Situation of the Liver.—The liver occupies the uppermost portion of the abdominal cavity, being placed immediately beneath the diaphragm, into the concavity of which, its upper surface fits. Three-fourths of the organ lie to the right of the median line, and one-fourth to the left. Its upper or convex surface reaches to the level of a plane drawn through the cartilage of the fifth rib in front and the ninth dorsal vertebra behind. At a point midway between the right parasternal and mammary lines it is immediately behind the fourth intercostal space, or three finger-breadths above the lower border of the right lung. The lower edge of the liver, starting from behind, extends outwards from the twelfth dorsal vertebra, along the eleventh rib, towards the axilla. "In the mammary line it lies immediately beneath the costal arch; while in the epigastrium, an average position for the lower margin is about half-way between the xyphoid cartilage and the umbilicus." (Bury.) The left lobe occupies the upper portion of the epigastrium, and lies beneath that part of the diaphragm upon which the heart rests. Its lower border, starting in the median line at the point above indicated, "passes obliquely upwards, to reach the left costal margin at the junction of the seventh and eighth costal cartilages; behind the cartilages it is continued as far as the heart, and terminates a little to the right of its apex." (Bury.)

Methods of Examining the Liver.—The methods employed for the physical examination of the liver include:

1. Inspection.
2. Palpation.
3. Percussion.
4. Auscultation.

1. **Inspection.**—Inspection of the region of the liver (the right hypochondrium being usually accepted as this place) demands full exposure of the lower portion of the thorax and the entire abdomen. While its utility is limited, nevertheless, if advantage is taken of all the possible information it affords, its practice is ofttimes capable of adding greatly to one's knowledge of the case in hand.

First note the presence or absence of any undue prominence in this location. In the adult, hepatic enlargements are rarely sufficiently great to assert themselves on inspection. In the case of children, in whom there is always considerable elasticity of the ribs, it is different, for in them an enlargement is not unlikely to exhibit itself as an undue prominence in the right hypochondrium. Even in adults, at times, a diffuse elevation of the right hypochondrium is observed in case of hepatic enlargements; the lower ribs may even be thrown outwards. When the liver is considerably enlarged and the abdominal walls are thin, it may be possible to observe the lower margin of the liver rising and falling with the respiratory movements. In the case of tumors extending below the costal margin, inspection readily recognizes their presence, owing to the non-resistant character of the abdominal walls.

Localized œdema over the right hypochondrium is suggestive of acute suppurative hepatitis. (Abscess.)

All prominences in the right hypochondrium must not, however, be associated with hepatic disorder, for this condition may be produced by right-sided pleurisy with effusion; but in the latter case the intercostal spaces bulge more or less prominently, thus differing from hepatic enlargements, some cases of abscess of the liver excepted.

Inspection also enables us at times to discover pulsations of the liver, although the presence of this symptom is more readily detected by palpation. Such pulsations indicate either tricuspid regurgitation or aneurysmal dilatation of the abdominal aorta. A diastolic pulsation of the liver may be observed in some cases of aortic insufficiency with cardiac hypertrophy. It is brought about by the extraordinary fullness and strong pulsations of the hepatic artery and its ramifications in the parenchyma of the liver.

The information obtained by inspection in liver disorders need not be confined to observation of the right hypochondrium. Disease of the liver is prone to interfere with the portal circulation, as a result of which, enlargements of the superficial abdominal veins may be noted. When the portal obstruction is extreme, there may form a great enlargement of the superficial veins of the abdomen centreing at the umbilicus, and sometimes spoken of as the "*caput medusæ*." This condition may also result from obstruction of the descending vena cava. When the obstruction is portal, both the superior and inferior epigastric veins are simultaneously enlarged; when the obstruction is in the descending vena cava, the inferior epigastric veins are the first to undergo enlargement.

Occasionally, pulsations of the superficial abdominal veins may be observed as an accompaniment of cirrhosis of the liver.

2. **Palpation.**—Palpation gives information only in cases in which the liver is enlarged. It should be practised as follows: The patient should be in a reclining posture, with his head and shoulders raised somewhat, and his thighs flexed upon his abdomen. Care should be observed that the abdominal walls are thoroughly relaxed. It is next in order to feel for the lower edge of the liver. In the healthy subject this is not palpable, as it

does not lie below the right costal arch. The tips of the palmar surface of the fingers should be pressed firmly inwards below the margin of the ribs, during or at the end of expiration. In this way it is possible to get the fingers well beneath the ribs, so that when inspiration takes place, the liver is forced downwards, and its lower edge is felt to glide beneath the examining fingers.

Another method of palpating the liver, though not as generally useful as the one above described, may be employed when the patient's abdominal walls do not relax sufficiently. It consists in palpating the liver with the patient in a sitting position, and the body bent well forward.

In every case, palpation should be performed at different levels before deciding that the edge of the liver cannot be felt.

Palpation of the liver in the epigastric region is not a difficult matter, the lower edge here being felt at a point midway between the ensiform cartilage and the umbilicus.

Glenard has devised what he has been pleased to call the **"thumb method"** of palpating the liver. While it may be useful in some cases, its routine employment to the exclusion of the method above described is not to be recommended. Pressure is made in the right lumbar region with the four fingers of the left hand. The right hand makes pressure from below upwards, starting in the right groin. The thumbs of both hands are free, and with them the examiner endeavors to discern the margin of the liver. The patient should be instructed to take deep inspirations, so as to depress the organ to the maximum extent.

Combined palpation is recommended by Semmola and Gioffredi as adapted to cases in which the abdomen is distended with fluid or gas. "With the left hand open and applied against the lumbar region, pressure is made upwards in the attempt to raise the liver and bring it closer to the abdominal wall, while the right hand makes gentle but continuous pressure with its ulnar border, and gradually works down so as to reach the border of the enlarged liver."

"Dipping for the liver" may also be employed as a means of palpation in cases of enlargement of that organ associated with ascites or tympanites. The finger-tips are held perpendicularly to the abdominal surface over the point where the liver is expected to be. They are then pushed suddenly into the abdominal walls. The fluid or gas, as the case may be, is thus pushed aside, and the fingers come in contact with the liver surface. By repeating the manœuvre over different points it is possible to locate the hepatic margin. While this method gives valuable information, it is not without danger in practice, especially in cases of abscess, hydatid cysts and distended gall-bladder. Semmola and Gioffredi's combined method is safer, and nearly as reliable.

The information obtainable by palpation includes the recognition of downward displacements and enlargements of the liver, the character of the liver surface, its consistence, the movements of the organ during respiration, and the presence or absence of friction fremitus and hydatid thrills.

(*a*) **Downward Displacements and Enlargements of the Liver.**—So far as palpation is concerned, these cannot be differentiated. For this purpose one must employ percussion, which will enable us to determine the upper boundary of the liver. If this is in its normal position, then the downward extension must be an enlargement; whereas if it occupies a lower position than it should, there must be a displacement. The conditions giving rise to enlargements and displacements will be mentioned shortly, when treating of percussion.

(*b*) **The Character of the Liver Surface.**—In health this is smooth. Unless the alterations of the surface are very obtrusive or the abdominal walls thin, changes are not discernible. Especially does this remark apply to the discovery of the granular surface found post-mortem in cases of cirrhosis of the liver. I doubt very much if this sign is often available for diagnosis, and the same may be said of the roughening observed in some cases of tubercular peritonitis. More pronounced alterations in the liver surface may, however, be felt. Thus, nodular formations are palpable in cases of cancer and gummata. In cancer we find the nodules large, hard, sometimes resembling ridges, and with cup-shaped central depressions. The gummatous elevations are smoother and flatter than those of cancer. An additional differential sign is the progressive enlargement of the liver in cancer, and this is rarely observed in syphilitic disease. The history of the case is also important, for cancer of the liver is nearly always secondary to cancer elsewhere, and in the case of gummata we may obtain a history of syphilitic infection or evidences of the ravages of that disease in other organs or tissues. Abscesses of the liver and hydatid cysts containing fluid give round, smooth swellings, fluctuating to the touch.

(*c*) **The Consistence of the Liver.**—This will be found to be greatly increased in cancer; indeed, in this disease it has been described as of a stony hardness. An increased consistence is also observed in hyperæmia, cirrhosis, and amyloid disease. In the latter, the organ may at times be quite hard. A soft and flabby consistence is noted in fatty degeneration of the liver. A sense of fluctuation occurs in abscess and cystic tumors.

Information is obtainable from the character of the edge of the liver. In amyloid disease it is rounded; in interstitial hepatitis and hypertrophic cirrhosis it is sharper than normal.

(*d*) **Mobility During Respiration.**—An important means of recognizing enlargements as belonging to the liver lies in the fact that they move about half an inch with respiration. The exceptions to this are as follows:

i. When the movements of the diaphragm are slight or absent.

ii. When there are adhesions of the liver to the abdominal walls or spinal column.

iii. When there are marked displacements of the liver as the result of rupture or relaxation of the suspensory ligament.

iv. When a tumor of the liver has attained such a great volume as to press up into the thoracic cavity.

v. In cases of great abdominal distention by fluid or gas.

(*e*) **Friction Fremitus and Hydatid Thrill.**—Friction fremitus occurs over the liver when there is roughening of the portion of the peritoneum that comes in contact with the liver. It gives a sensation as of the creaking of leather. It may be elicited by pushing the abdominal walls over the surface of the liver. It is also evident on auscultation. It indicates a past or present peritonitis. The history of the case will decide.

The *hydatid thrill* is a peculiar tremulous sensation imparted to the hand laid flat on the hydatid cyst, when percussed with the fingers of the other hand. The same sensation may be observed in other cystic growths; hence too much reliance must not be placed upon it.

Palpation of the Gall-Bladder.—It is safe to assume, as a general rule, that the gall-bladder is not palpable in healthy subjects. When, however, it is somewhat distended, the subject possessed of thin abdominal walls, and the stomach and bowels empty, it may be felt as a smooth, round, fluctuating tumor. It has even been said that it may be discerned by inspection, under such circumstances. The position of the gall-bladder is immediately beneath that point at which a line drawn from the right acromion process to the umbilicus crosses the right costal arch.

When the cystic or common duct is obstructed and the secretions are dammed up, the gall-bladder undergoes great distention. It then becomes apparent to both inspection and palpation, being manifest as a pear-shaped tumor moving with the liver during respiration. The gall-bladder may also be enlarged by inflammatory exudate or by accumulation of gall-stones. When the enlargement is due to malignant disease or inflammatory thickening, the tumor presents a dense irregular sensation to the examining fingers. In the case of the presence of calculi it may be possible to perceive a grating sensation, from the rubbing of one calculus against another.

3. **Percussion.**—When practising percussion in the examination of the liver, it must be borne in mind that there is an area under which the liver substance comes in direct contact with the abdominal walls, and over which absolute dullness or flatness is obtainable. This is known as the area of absolute dullness. It may best be elicited by light percussion. There is a second area in which the liver substance is separated from the overlying surface by lung tissue. This is known as the area of relative dullness. Light percussion in this locality gives the pulmonary resonance,—modified,

of course, by the existence of a solid organ beneath. Deep percussion elicits the relative dullness from that organ. The following are the outlines of normal absolute liver dullness:

	Upper Boundary.	Lower Boundary.
In middle line, .	Base of ensiform cartilage.	Nearly half-way between ensiform cartilage and umbilicus.
In mammary line,	6th rib.	Costal margin.
In axillary line, .	8th rib.	10th rib.
In scapular line, .	10th rib.	11th rib.
Near the spine, .	11th rib.	

The area of relative dullness lies above that of absolute dullness. Its upper boundary runs parallel to and about one and a quarter inches above the upper line of absolute dullness. Its upper limit will be found to be at about the fifth interspace, or a little below the highest point of the liver. It is very difficult to elicit posteriorly. Fortunately this is not of much clinical importance, as all the necessary information may be obtained from in front.

Various difficulties may be encountered when endeavoring to map the outlines of the liver by percussion. The lower limit behind cannot be accurately delimited because of the merging of the dullness of the right kidney and the lumbar muscles with that of the liver. Care is required when delimiting the lower margin of the liver median line in front, because its thin edge overlaps the stomach and transverse colon. Strong percussion carelessly performed fails to recognize the thin hepatic tissue, owing to the tympanitic resonance from the parts beneath. Then, too, the character of the percussion-note must vary according to the state of tension of the abdominal walls and the degree of distention of the stomach and intestines with gas. Difficulty in delimiting the left lobe of the liver arises from the merging of the liver into the cardiac dullness.

We are now prepared to study the significance of alterations in the areas of liver dullness. These include diminished or increased liver dullness.

Diminished liver dullness may be due to diminution in the size of the liver itself, or to the separation of the liver from the abdominal walls by the interposition of distended stomach or intestines. Pulmonary emphysema likewise renders the area of superficial dullness less extensive, and may even lessen the absolute area. In some cases it makes the superficial dullness larger at the expense of the relative area. When the emphysema is quite marked, it may obliterate the relative dullness altogether, leaving the absolute dullness of normal size, though lower in position.

An apparent diminution in the area of hepatic dullness occurs in those cases in which the lower line of dullness is raised by the pressure of tumors and fluid accumulations from below. In this case, however, there will be an elevation of the upper line of relative dullness, owing to the displacement of the liver upwards.

The diseases of the liver of which diminished area of dullness is characteristic include :

a. Cirrhosis of the liver.
b. Acute yellow atrophy of the liver.

(*a*) **Cirrhosis of the Liver.**

A variety of types of cirrhosis of the liver have been described. In some of these the liver is greatly increased in size, while in others the reverse condition obtains. The clinical classification of Osler is undoubtedly the best adapted for practical work, including (i) the alcoholic ; (ii) the hypertrophic cirrhosis of Hanot ; (iii) syphilitic cirrhosis ; (iv) capsular cirrhosis or chronic perihepatitis. In the first and fourth varieties the liver is diminished in size, and these concern us most at the present moment.

In the diagnosis of atrophic cirrhosis of the liver the history of alcoholic indulgence covering a term of years is of the highest importance, the liquors which seem to be especially active in this connection being whiskey and brandy. Excessive indulgence in beer inclines to the production of cirrhosis, with fatty degeneration. Alcoholic indulgence is probably more frequent than all other causes combined. There is some reason to believe that cirrhosis of the liver may follow certain infectious diseases, notably scarlatina and malaria.

The symptomatic picture in hepatic cirrhosis varies greatly. In some instances there are absolutely no symptoms. The explanation accorded such cases is that the collateral circulation has been established. When symptoms are present, they may be attributed to two influences, namely, the obstruction of the portal circulation and a toxæmia, the true nature of which has not yet been established. The obstructive symptoms refer to the impeded portal circulation. Chronic gastric catarrh and its attendant clinical phenomena result from congestion of the stomach. Ascites is, however, the most prominent of the portal obstructions. This may cause remarkable abdominal distention. In the effort to establish a collateral circulation, certain veins become dilated. The most important of these, from a clinical standpoint, are those at the lower end of the œsophagus, which may become of enormous size, and sometimes rupture, leading to severe hæmatemesis. Enlarged veins may be seen on the abdomen. At times they may even be varicose, in extreme cases presenting a large bunch about the umbilicus, and known as the *caput medusæ*. Intestinal hæmorrhage is sometimes observed. Jaundice is not a common symptom, and, when present, it is of but slight degree. The urine is usually reduced in quantity. The spleen is gradually enlarged, even to two or three times the normal. Hæmorrhoids are nearly always present.

Toxic symptoms, other than those dependent upon the gastro-intestinal catarrh, do not set in until late. They include, for the most part, cerebral phenomena, as delirium, stupor, coma, or convulsions.

In the first stage of the disease the liver is enlarged. Later it becomes contracted, as already stated.

Capsular Cirrhosis.—In this variety of liver disease there is an enormous thickening of the capsule of the liver, with associated contraction of the organ. According to Fagge and White, to whose labors we owe most of our knowledge of the disease, the liver is "never markedly cirrhotic; its tissue was nearly always soft." It is usually found associated with a chronic inflammation of the capsule of the spleen, a chronic proliferative peritonitis, and granular kidneys. Able clinicians seem to have been unable to diagnose the condition during life. According to White, the important diagnostic points are the symptoms of atrophic cirrhosis as above described, interstitial nephritis, recurrent ascites, and the absence of jaundice. In another class of cases there are also the signs of perihepatitis, perisplenitis, proliferative peritonitis, adherent pericardium, and chronic mediastinitis.

(*b*) Acute Yellow Atrophy of the Liver.

This is an exceedingly rare disease, characterized at its onset by the symptoms of a gastro-duodenal catarrh, with some jaundice. After a variable period—from a few days to a couple of weeks—there appear symptoms indicative of the serious nature of the case. These include headache, delirium, convulsions, vomiting, subcutaneous or submucous hæmorrhages, and rapid increase of the jaundice. Finally coma appears, and increases until death. The urine is found to contain bile, tube-casts, and at times leucin and tyrosin. The prognosis of acute yellow atrophy of the liver is absolutely unfavorable. Its diagnosis depends upon the severe and rapidly-increasing jaundice, associated with marked diminution in the size of the liver, delirium, and the leucin and tyrosin in the urine.

Increase of the liver dullness can take place only from increase in the size of the liver. An apparent increase of the liver dullness may result from the contact of the organ with normally or pathologically solid structures. For example, in right-sided pleurisy, with effusion, the liver dullness will merge with that of the effusion. A tumor or abscess between the liver and diaphragm will produce an increase of dullness upwards. Inflammation of the lower lobe of the right lung (pneumonia) will produce a similar result. An apparent increase of dullness downwards may result from distention of the stomach and colon by solid materials, malignant growths of adjacent structures, and distended gall-bladder.

The indications afforded by extension of the lines of liver dullness have been epitomized by Bury as follows:

The upper limit of dullness being higher than normal, the lower edge of the liver occupying the normal position, the condition must be—

(*a*) Enlargement of the liver upwards;
(*b*) Moderate pleuritic effusion, the dullness of which merges with that of the liver; or,
(*c*) The liver is enlarged and pushed upwards, as by ascites.

The lower line of dullness being normal, and the upper limit lowered, there is suggested

A moderate degree of emphysema.

When the lower edge of the liver is lower than normal and the upper limit is normal,

The liver must be enlarged.

If, with lowering of the lower limit of hepatic dullness, there is upward extension of the upper line of dullness, then—

(*a*) The liver may be enormously enlarged.
(*b*) There may be pleuritic effusion; or,
(*c*) There may be a large swelling or tumor between the liver and the diaphragm.

When the upper line of dullness is lower than normal, and the lower limit is lower than normal, there may be—

(*a*) Considerable emphysema; or,
(*b*) Pneumothorax.

When both upper and lower limits of dullness are higher than normal,

The liver is dislocated upwards.

When the upper limit of dullness is normal and the lower limit is higher than normal, it may be that—

(*a*) The liver is smaller than natural; or,
(*b*) There is ascites, tympanites or gas in the peritoneal cavity.

If no liver dullness can be detected, there is—

(*a*) Much ascites,
(*b*) Distention of the intestines by gas,
(*c*) Extreme atrophy of the liver, or
(*d*) The liver occupies an abnormal position, owing either to its mobility or to congenital malposition.

Attention to the data in the above schema will enable the student to differentiate enlargements from displacements, and also to determine the presence or absence of conditions simulating these lesions. It also gives the necessary data for determining the presence of atrophies and conditions simulating the same. It is next in order to give in detail the causes and conditions producing enlargements and displacements, and to describe the clinical phenomena by which each of them may be recognized.

Displacements of the liver may result from—

a. Rachitic chest.

b. Tight lacing.
c. Pleuritic effusion.
d. Pneumothorax.
e. Croupous pneumonia.
f. Subphrenic abscess.
g. Tumors.
h. Wandering liver.
i. Pressure of tumors and effusion from below.

Enlargements of the liver may be—

1. Diffuse, *i.e.*, involve the entire organ; or,
2. Circumscribed.

Circumscribed enlargements of the liver may be due to—

a. Cancer and other tumors of the liver.
b. Abscess of the liver.
c. Hydatid cysts.

Diffuse enlargements of the liver may be due to

a. Hyperæmia.
b. Inflammation.
c. Hypertrophic cirrhosis.
d. Fatty degeneration.
e. Amyloid degeneration.

Of the above-mentioned enlargements of the liver, the following are **accompanied by pain**:

a. Hyperæmia.
b. Inflammation.
c. Hypertrophic cirrhosis.
d. Tumors, especially those of malignant character.
e. Catarrh or obstruction of the gall-duct.
f. Abscess of the liver.

And the following are **painless**:

a. Fatty liver.
b. Amyloid degeneration of the liver.
c. Hydatid cysts.

Displacements of the Liver.

(*a*) **From Rachitic Chest.**—As described elsewhere, the rachitic chest is characterized by a lateral sinking-in of the middle ribs and projection of the sternum (pigeon-breast), with arching backwards of the sternum. The capacity of the thorax is lessened; hence the diaphragm is depressed, and, with it, the liver takes a lower position. The diagnosis is based upon the recognition of the rachitic type of chest. The exact state of affairs may not be recognized, because many cases of rachitis are associated with enlargement of the liver, in addition to the displacement.

(*b*) **Tight Lacing.**—The effect of tight lacing is to force the liver to take a position lower than the normal. When the practice has been of long standing and extreme, the organ may also be deformed. It is sometimes possible, in the latter event, to feel a furrow, and below it the right lobe of the liver extending downwards, even to the anterior superior spinous process of the ileum. The gall-bladder also takes a much lower position than normal, and inflammation or gall-stones may then offer a very interesting diagnostic problem. It is not always easy to determine the true state of affairs, for the depressed lobe of the liver is thin and movable. Sometimes its nature is still more obscured by coils of intestines lying in the furrow and separating the supposed tumor from the liver. Under such circumstances, the only sign or symptom to put the physician on his guard is the outward evidence of tight lacing.

(*c*) **Pleuritic Effusion.**—The position of the liver in right-sided pleurisy with effusion is our main guide as to the extent to which the diaphragm is depressed. When the effusion is large, the lower margin of the liver may be on a level with the umbilicus. This displacement, however, is not manifested equally over the entire liver, the left lobe being relatively higher than the right. When the effusion is moderate, it sometimes happens that the downward pressure is exerted mostly posteriorly. This causes a tilting of the liver, in which case the lower line of dullness anteriorly may be higher than normal.

(*d*) **Pneumothorax.**—The degree of hepatic displacement from pneumothorax may be extreme; indeed, the upper convexity of the liver may appear below the ribs. The history of the case, the remarkable physical signs of air or gas in the pleural cavity, and the displacement of the heart, make the diagnosis certain.

(*e*) **Croupous Pneumonia.**—The displacement of the liver from this cause is neither great nor important, from a diagnostic standpoint.

(*f*) **Subphrenic Abscess.**—As is the case with many other disorders, the history of a cause capable of giving rise to this condition is an important factor in the diagnosis of subphrenic abscess, or, as it is sometimes called, *subphrenic peritonitis.* These causes include perforating ulcer of the stomach or duodenum, appendicitis, renal abscess, cholecystitis, perforation of the intestine, and pelvic suppuration. The size which these abscesses may attain is sometimes enormous. They are thus perfectly capable of depressing the liver and crowding the diaphragm upwards. They sometimes perforate the diaphragm, producing empyema. Sometimes they contain gas, because of their communication with gas-containing organs.

The symptoms of subphrenic abscess are a direct continuation of those of the lesions which give rise to it, there being only the intervention of the especial phenomena attendant upon the perforation. We find the ordinary symptoms of suppuration, *i.e.*, septic fever with its chills, irregular fluctuations of temperature, and sweats, and the physical signs. The conditions with which subphrenic abscess may be confounded are empyema and abscess of the liver. Curtis very graphically gives the following differentiation of subphrenic abscess and empyema : " In empyema the upper surface of the fluid will describe a curved line on the side of the chest, with its concavity turned upwards. In subphrenic abscess the upper limit of the fluid will also be a curved line, but the convexity will look upwards, the pus being contained beneath the dome of the diaphragm. If the aspirating needle be inserted, the flow of the fluid from it will be reversed in the two cases, the flow being the strongest in empyema on expiration and in subphrenic abscess on inspiration, the pressure of the diaphragm making the difference. If gas only is contained in the cavity which the needle enters, its passage in and out will show the same peculiarities, and can be demonstrated by connecting the needle with a manometric tube, or by holding a lighted match where the current can detect the flame."

(*g*) **Tumors.**—Tumors produce displacement of the liver by reason of their weight and mechanical action. They will be referred to shortly under the heading of Enlargements.

(*h*) **Wandering Liver ; Floating Liver ; Hepoptosis.**—The majority of cases of movable liver occur in women, tight lacing and pregnancy being the most prolific causes of the trouble. It is nearly always attended by flaccidity of the abdominal walls, and yet cases have been observed in persons whose abdominal walls were not relaxed in the slightest degree. It has been suggested with good reason that the causes named are active only when there is a congenital anomaly of the ligaments of the liver, either in the direction of unusual length or undue extensibility. It is not unusual to find cases of hepoptosis associated with displacements of other abdominal viscera, notably of the kidneys ; and the suggestion made by Kellogg that many cases of prolapsus uteri are associated with a similar condition of many abdominal organs is entitled to the greatest respect.

In some few cases floating liver gives rise to no symptoms whatever, the condition being discovered accidentally in the course of an examination for some other complaint. Usually, however, it is associated with most distressing sensations, especially when it is not the only viscus displaced. The subjective symptoms include a sense of dragging or weight in the epigastrium and right hypochondrium, and this may become so severe as to amount to a veritable neuralgia. As a rule, the pain or discomfort is confined to the region of the liver, but it may radiate to distant parts, especially to the right shoulder. It is, as a rule, intensified when the patient is in an erect position. In still other cases the malposition of the liver leads to torsion of the ductus choledochus. As a result, we have obstructive jaundice and all the phenomena of hepatic colic. Even the portal vein may be compressed or subjected to undue tension,—in which case portal stasis ensues, and ascites appears. The diagnosis of cases of this character is not always easy. It is made, however, by the recognition of a tumor having the contour of the liver, and this tumor is freely movable. At the same time, physical examination shows that the liver is absent from is normal location.

(*i*) **Pressure of Tumors and Effusion from Below.**—Under these circumstances the liver is pushed upwards. The diagnosis is based upon the recognition of the primary condition. For further study of this subject the reader is referred to the chapter on Abdominal Enlargements.

I. Diffuse Enlargements of the Liver.

(*a*) **Hyperæmia of the Liver.**—Hyperæmia of the liver occurs in two varieties, namely, the active and the passive. (i) *Active hyperæmia of the liver* is an aggravation of the normal hyperæmia of the liver following the ingestion of food, and is due to indulgence in an excessive quantity of food and drink. It may also constitute one of the phenomena of acute febrile disorders. Its symptoms are not by any means characteristic. They include a sense of discomfort in the hypochondrium, and slight enlargement of the organ. (ii) *Passive hyperæmia of the liver* is always a secondary disorder, and is dependent upon local or general circulatory disturbances. Of the local causes, pressure upon the hepatic vein by a tumor and obstruction by thrombi are the most important. It is also claimed that tight lacing may bring about a localized hyperæmia of this character. Of the general circulatory disturbances giving rise to this condition, that produced by weak or dilated heart or uncompensated valvular lesions is the most important. It may also be observed in emphysema, cirrhosis of the lungs (fibroid pneumonia) and intrathoracic tumor. The symptoms arising from this condition are always vague; indeed it is difficult, if not actually impossible, to separate them from those of the primary disease. Naturally the patient suffers from the various phenomena of chronic gastric catarrh, but this pathological change is not infrequently the direct result of heart disease itself. There may be hæmatemesis. Dropsy is not uncommon, and it may be that ascites appears prior to the onset of the generalized œdema. Slight jaundice, clay-colored stool and biliuria may be present. The diagnosis of passive hyperæmia of the liver is based upon the physical signs of a generalized enlargement of the organ, together with the presence of the symptoms and signs of the lesions capable of giving rise to this condition. The extent to which the liver may enlarge as a result of passive hyperæmia is sometimes astonishingly great, it not being impossible to find the lower margin of the organ a full hand-breadth below the normal line of dullness. Jaundice of slight grade is sometimes present, there being only a yellowish tinting of the conjunctiva and sallowness of complexion. When, as in cases of organic heart disease, there is also cyanosis, there results a greenish tint of the skin. When passive congestion of the liver is of long standing there ensues cachexia, with anæmia, poor circulation, and general nervous depression.

(*b*) **Hypertrophic Cirrhosis.**—Aside from the enlarged liver which characterizes the early stages of atropic cirrhosis, there is a special variety known as the hypertrophic cirrhosis of Hanot. The vast majority of cases occur in male subjects, and a fair number in children. There is absolutely nothing known concerning its etiology. It is a disease running a very chronic course, and is characterized by jaundice, which, however, is usually but slight in intensity; biliuria, and more or less severe pain in the region of the liver, associated with nausea and vomiting. The liver and spleen are enlarged. Ascites and dilatation of the superficial abdominal veins are absent. A hæmorrhagic tendency is a remarkable feature of some cases. Fever of mild degree is often present, and is apt to become intensified during the periods of exacerbation of pains.

Notwithstanding its mildness in the majority of cases, the jaundice assumes a prominent part in the symptomatology of the disease. It is not uncommon for it to be the first symptom to suggest to the patient the fact that he is ill. It may come on gradually or suddenly,—usually the former. Its association with gastro-enteric disturbance ofttimes leads it to be mistaken for catarrhal jaundice. It is accompanied by slight fever and some hepatic enlargement. The jaundice disappears under appropriate treatment; but the liver, while diminishing in size, never regains its normal proportion. Later, jaundice of slight degree becomes a permanent feature. At the same time the patient is liable to attacks of *icterus gravis*, with high fever and delirium, and in which he may die. In some cases the jaundice shows no amelioration, gradually becoming more intense until death.

The hypertrophy of the liver is surely progressive, although there may be periods during which it diminishes somewhat in size. The organ may attain an enormous size, *i.e.*, its lower margin may reach three or four finger-breadths below the normal line of dullness. Its surface is ordinarily smooth to palpation; its consistence is increased, though not to the extent of a stony hardness. The perihepatic friction-rub is sometimes present.

Pain may be spontaneous or produced by manipulation of the hepatic region, as in examination. It is general throughout the region of the liver.

The enlargement of the spleen is sufficient to make that organ recognizable by palpation. Its surface is smooth, and the organ is painful to manipulation.

As the disease progresses, evidences of malnutrition appear. The abdomen alone remains of large size, the enlargement being especially noticeable in the epigastric and right hypochondriac regions. Hæmorrhages from the mucous membranes and skin take place. The cause of this symptom is believed to be an acquired hæmophilia. The cardio-vascular system fails. Finally death ensues.

(*c*) **Fatty Degeneration of the Liver.**—Two varieties of fatty change in the liver have been recognized, namely, fatty infiltration and fatty degeneration. The former is but part of a general obesity. It is especially apt to occur in persons who subsist largely upon a diet of fats and carbohydrates and take insufficient exercise. It may also occur as one of the phenomena of the various anæmias, phthisis, cancer, syphilis, and gastro-intestinal disorder. True fatty degeneration of the liver results from poisoning by phosphorus and alcohol mainly, although it has been produced by arsenic, antimony and carbonic oxide. Cases have also been observed secondary to other liver disorders.

The symptoms of fatty liver are by no means sufficiently pronounced in most cases to direct attention to that organ. In the majority of cases they are masked by those of the primary disorder. Inasmuch as the latter is usually of a character calling for a scant diet, and that of easily-digested food, very little demand is made upon the liver; hence its functional weakness does not become apparent unless the degenerative changes are extreme. In some cases the great enlargement of the liver may, by exercising considerable tension upon the capsule of the organ, produce pain or sense of stretching, thus directing attention to the part. Examination then shows the liver dullness to extend several finger-breadths below the costal margin. Palpation shows the surface of the liver to be smooth, and its consistence below the normal standard. Not infrequently the great thickness of the abdominal walls may cause the results from physical examination to be very unsatisfactory. Jaundice is usually absent. There is no ascites or other evidence of interference with the portal circulation. The spleen is not enlarged.

(*d*) **Amyloid Degeneration of the Liver.**—This is never a primary affection. Hence the recognition of the presence of the various pathological conditions from which it may result constitutes an important element in its diagnosis. These conditions include, in a general way, "profound exhaustion attendant upon the various cachexiæ." Especially does amyloid liver follow prolonged suppuration. The diseases which have, with good reason, been credited as its cause include pulmonary tuberculosis, suppuration of bones, syphilis, malaria, osteomalacia, leukæmia, pseudoleukæmia, malignant tumors, and Bright's disease.

Aside from the etiological factors above mentioned, the diagnosis rests upon the recognition of great enlargement of the liver. Inspection may show an unnatural prominence in the right hypochondrium and the epigastrium. The hepatic enlargement is uniform, its surface is of almost stony hardness, and its free edge is rounded. There are commonly associated similar changes in the spleen, kidneys and intestines. The etiological factors of amyloid degeneration are, many of them, the same as those causing fatty liver. The stony consistence of the liver and the roughness of its surface in amyloid degeneration serve for a differentiation. Intestinal disorders, especially diarrhœa, are not uncommon. The patient becomes cachectic, and dies.

II. Circumscribed Enlargements of the Liver.

(*a*) **Carcinoma of the Liver.**—Cancer of the liver may be either primary or secondary,—the latter being frequent, the former exceedingly rare. The etiology of the primary cases is that of carcinoma in general. The secondary cases occur either as a concomitant or as a sequel of malignant disease in almost any portion of the body. In many instances, however, the clinical evidence of the primary lesion is so vague that it is not recognized during life. Then, on the other hand, carcinoma of the liver is very prone to occur as a complication of the same lesion in other situations, it being stated authoritatively that fully 50 per cent. of cases of carcinoma of different organs coming to autopsy show secondary formations in the liver.

The clinical picture of carcinoma of the liver presents the widest possible variations. Sometimes the disease runs a latent course, its true nature not being discovered until a progressive weakness and emaciation leads to a thorough physical examination of the patient. In others, the presence of malignant disease of the liver is overlooked because of the known existence of carcinoma in other situations.

The first symptoms observed are usually those referred to the gastro-intestinal tract. They consist of loss of appetite, sensation of weight in the epigastrium, nausea and vomiting, and constipation. The urinary changes are important. The urine is greatly diminished in quantity, and contains a very small percentage of urea. Urobilin and albumin are also found in the urine.

The physical signs are those of an enormously and irregularly-enlarged liver. Palpation shows the lower edge of the liver to be four or five inches below the costal arch. It may even extend below the crest of the ilium. The irregular or umbilicated nodules over the surface of the organ are readily palpable. The substance of the liver is very hard. Percussion enables us to confirm the results of palpation, and, furthermore, to delimit the upper limits of the liver. As a rule, the cancerous enlargement extends throughout the entire organ, but one portion or lobe is more markedly affected than others. When the patient is greatly emaciated, as is not unusual in the late stages, it may be possible to recognize the liver contour and its cancerous nodules by inspection.

Jaundice is a very common phenomenon, and when once pronounced is progressive. It is due to the pressure of cancerous nodules upon the hepatic or common bile-duct.

Ascites may also occur, but is not observed as frequently as is jaundice. It is said that in this disease it is produced by the peritoneal irritation arising from the carcinomatous nodules. It is reasonable to believe that it may also depend upon obstruction of the portal system. Sooner or later the patient exhibits the distinctive features of the cancerous cachexia. This, in conjunction with the characteristic enlargement, establishes the diagnosis.

The conditions with which carcinoma of the liver may be confounded include certain cases of hepatic syphilis, amyloid degeneration, and hypertrophic cirrhosis. Gummatous formations in the liver may closely simulate cancerous nodules, but the latter are harder than the former. Cancer runs a much more rapid course than syphilis, the patient dying within fifteen months at the outside. Treatment produces good results in hepatic syphilis, but is ineffectual in carcinoma.

Pain is a very variable factor. Usually it is present and of a high grade. Sometimes it amounts to nothing more than a sense of weight or fullness in the right hypochondrium, which is not surprising, in view of the size and weight of the hepatic tumor. The pain seems to bear a direct relation to the extent to which the peritoneum is involved by the malignant changes.

In amyloid degeneration we have the etiological factors to guide us; also the smooth surface of the liver, the associated splenic enlargement, and renal disease.

Certain rare types of malignant disease of the liver may give rise to considerable diagnostic difficulties. Thus in the so-called *cancer with cirrhosis*, the liver is but slightly enlarged; indeed, it is stated by some authorities that it may even be diminished in size. Whether the clinical recognition of these cases is possible is doubtful.

(*b*) **Abscess of the Liver.**—Abscess of the liver being practically always a secondary affection, its etiology becomes an important part in its diagnosis. Two general varieties have been recognized,—one the large, single collection of pus, and the other multiple, and dependent upon pyæmia. The former includes the cases usually designated as tropical abscess and those dependent upon traumatism. The tropical abscess may apparently be idiopathic, but seems to be a sequela of dysentery, its incidence following this disease being attributed to infection with the *amœba coli.* In cases which give no history of dysentery, the amœbæ coli have nevertheless been found in the stools. Predisposing factors include residence of the unacclimated in a tropical climate, indulgence in overeating and drinking, exposure to bad hygiene, and hardships. In some cases it seems to have resulted from ulceration and other intestinal affections, typhoid fever, and appendicitis.

When hepatic suppuration is the result of pyæmic infection, numerous purulent foci will be found post-mortem. These will be scattered irregularly throughout the entire organ, and will be associated with similar collections in other portions of the body, excepting in those cases in which the primary suppuration is situated within the portal circuit. Some cases originate in septic disease in or about the rectum, or after surgical operations on that portion of the body.

Rare causes of abscess of the liver are found in local tuberculosis, either of the liver itself or of the bile-ducts, suppurative changes in hydatid cysts, the presence of ascarides and other parasites in the bile-ducts, and the penetration of the liver by foreign bodies, as fish-bones, needles, etc.

Tropical abscess of the liver is usually single. It involves the right lobe in the great majority of cases, and may attain an enormous size. Ordinarily such abscess contains from three to eight quarts of pus.

The clinical phenomena of uncomplicated abscess of the liver are the constitutional symptoms of suppuration and the physical signs of a circumscribed enlargement of the liver,—associated, possibly, with the ordinary evidence of local inflammation and disturbance of the hepatic functions. With the advent of complications, *e.g.*, extension of suppuration to adjacent organs, new symptoms assert themselves.

The constitutional symptoms are those of suppuration generally, namely, irregularly intermittent or remittent fever,—the appearance of high fever being associated with chills or rigors, and its disappearance with profuse sweats. With the first onset of this fever, there is usually some pain or tenderness in the right hypochondrium. These symptoms increase in intensity until the clinical evidence of circumscribed enlargement of the liver is obtainable, after which the diagnosis becomes clear. In less acute cases the symptoms are of more gradual onset and of minor severity. Still other cases run a chronic course, usually presenting the symptoms above mentioned, but occasionally without any special phenomena to indicate the nature of the illness until the hepatic enlargement is well advanced.

The physical signs include those obtainable by inspection, palpation, and percussion. When the abscess is large there will be prominence of the right hypochondrium, with protrusion of the lower intercostal spaces. It may be that the lower border of the liver is visible. When the abscess is superficial, there may be a localized œdema, with redness. On palpation, we may sometimes detect the perihepatic friction-rub, but only when the capsule of the liver is inflamed. It also enables us to discover the enlargement of the liver, and its size and shape. It is often possible to detect a sense of fluctuation in the enlargement. By percussion we are enabled to outline both the superior and the inferior borders of the liver.

The pain of abscess of the liver is quite a variable factor. In some cases it is intense; in others hardly noticeable. It is always aggravated by the respiratory movements, and greatly interferes with breathing.

Ascites is very rare. When present, it is due either to compression of the portal vein or to coexistent peritoneal inflammation.

Jaundice is present in about one-fourth of the cases. It is very rarely severe, ordinarily amounting to only an icteric pallor.

Urinary examination gives important information. At first there is increase in urea percentage; but with destruction of a considerable portion of the liver substance, we have hypoazoturia. Semmola and Gioffredi regard this latter as a most important diagnostic symptom; for nearly always, in fevers, we expect an increased percentage of urea. In making the volumetric analysis for urea, it is important that the result be based upon the entire quantity passed in the 24 hours.

The conditions with which abscess of the liver may be confounded include malarial fever, pulmonary tuberculosis, suppurating echinococcus cysts, dilatation of the gall-bladder, and to suppuration in adjacent structures.

From *malarial fever*, it is to be differentiated by the regularity of the remissions and exacerbations of the fever, the detection of the malarial organism in the blood, and by the curative influence of quinine in that disease.

From *pulmonary tuberculosis*, by the presence of the tubercle bacillus in the sputum, the history of the case, and the physical signs of pulmonary disease.

From *suppurating echinococcus cysts*, by the history of the case; *i.e.*, the knowledge of the existence of a tumor prior to the constitutional manifestations of suppuration, and by aspiration and examination of the contained fluids.

From *hepatic intermittent fever due to gall-duct obstruction*, by the regularity of the fever, the paroxysms being separated by long intervals of apyrexia; by the presence of jaundice, which becomes more marked with each return of fever; by the long duration of the case (even six or seven years); and by the presence of pains indicative of gall-stone obstruction in that condition.

Liver abscess rupturing into the lung, with expectoration of the pus, is readily recognized by the anchovy-sauce color of the sputum and the discovery of the amœba coli when it is examined under the microscope.

Abscesses of the abdominal walls do not move with respiration, as do all liver enlargements.

4. **Auscultation.**—With very few exceptions, auscultation in the examination of the liver has more of technical than of clinical interest. Owing to the fact that sounds are transmitted best through solid media, it is possible by means of auscultation to outline the limits of the liver by the transmission of the heart sounds. Such a practice is by no means free from error,

however, because the cardiac sounds may be transmitted along the ribs. This objection does not apply to the auscultation of that portion of the liver which lies covered by the soft abdominal walls.

Auscultation over the liver may discover certain arterial murmurs produced by compression of the abdominal aorta, or arising from aneurysm of the aorta or of the hepatic artery.

A well-marked murmur may be heard in those cases of cirrhosis of the liver in which the obstructed portal circulation has been compensated.

The perihepatic friction-rub is, however, the most pronounced symptom elicited by auscultation. It is due to the friction of roughened peritoneal surfaces against each other. It may occur in association with any disease of the liver or of other organ capable of inflaming the peritoneum in this locality.

Table of Physical Signs of Tumors of the Liver that Do Not Feel Solid. (Fenwick.)

	Abscess.	Hydatids.	Enlarged Gall-Bladder.
Etiology	Dysentery. Tropics.	Tape-worm.	Biliary colic.
Pain	Severe.	None.	Slight.
Tenderness	Severe.	None.	Rare.
Temperature	Remittent fever.	Normal.	Usually normal.
Jaundice	Rare.	Rare.	Occasional.
Ascites	None.	None.	None.
Loss of flesh	Rapid.	None.	None.
Progress of disease	Rapid.	Slow.	Slow.
Character of tumor	Usually rounded.	Rounded, with thrill on percussion.	Pear-shaped, opposite right 9th cartilage.

Table of Symptoms and Physical Signs of the Chief Solid Enlargements of the Liver. (Fenwick.)

	Chronic Congestion.	Fatty Liver.	Cancer.
Etiology	Alcohol. Heart disease. Emphysema.	Alcohol. Phthisis and wasting diseases.	Usually secondary.
Pain	Slight.	None.	Severe.
Tenderness	Moderate.	None.	Generally moderate; may be severe.
Jaundice	Slight.	None.	Usually well-marked.
Ascites	Often present.	None.	Often present.
Loss of flesh.	None.	Moderate.	Severe.
Other organs affected.	Heart and lungs often diseased.	Phthisis and changes in other organs.	Cancer elsewhere.
Progress of disease	Slow.	Slow.	Rapid.
Shape of liver	Regular.	Regular.	Irregular.
Surface	Smooth and firm.	Smooth and soft.	Hard and nodular.
Spleen	Sometimes enlarged.	Not enlarged.	Not enlarged.

Pain in Diseases of the Liver.—Pain rarely occurs in diseases of the liver unless the lesion is so situated as to irritate or stretch its peritoneal covering. The commonest variety experienced is a dull, heavy sensation in the right hypochondrium. Another characteristic pain of liver disease is one situated at the inferior angle of the right scapula. The production of pain by tension of the peritoneal covering of the liver depends in great measure upon the rapidity with which the enlargement takes place. Hence in acute congestion in which the swelling is usually not great, but takes place rapidly, pain is commonly present, while in amyloid degeneration, in which the enlargement is of slow progress, pain is absent. In hydatid disease pain may be either absent or present according as the growth is slow or rapid. In acute inflammation, or perihepatitis, pain is a prominent feature, owing to the involvement of the capsule of the liver in the inflammation. In syphilitic disease and cancer, the severity of the pain depends largely upon the involvement of the peritoneum. In the latter affection it may be quite severe. The most agonizing pains of liver disease, however, are those dependent upon obstruction of the gall-duct by a calculus, and generally known as gall-stone or biliary colic. It may also result from obstruction of the bile-ducts by ulceration or catarrhal inflammation. Biliary colic may be simulated by painful paroxysms dependent upon the pressure of an aneurysm of the hepatic artery.

Diseases of the Gall-Bladder and Bile-Ducts.

Acute and chronic catarrhal angiocholitis will be described in the pages devoted to the clinical study of jaundice.

1. Suppurative and Ulcerative Angiocholitis.

This disease is, as a rule, associated with similar changes in the gall-bladder. It usually finds its origin in infection, the predisposition to which has been established by the changes set up in the biliary structures by the presence of gall-stones. Occasionally it follows cancer of the duct and the presence of foreign bodies in the same. In the diagnosis of this disease, a history of the causes capable of giving rise to it, in connection with a septic type of fever, swelling and tenderness in the neighborhood of the liver, and enlargement of the gall-bladder, are the important conditions. Jaundice is usually present, but it is very variable in intensity.

2. Acute Infectious Cholecystitis.

This disease must be the result of infection of the gall-bladder. The micro-organisms known to have produced it include the colon bacillus, the bacillus typhosus, the pneumococcus, streptococci and staphylococci. Acute infectious cholecystitis is attended with more than usual interest because of the *comparative* frequency with which it is dependent upon typhoid fever. It is a disease of great fatality, but is fortunately of rare occurrence.

The diagnosis of infectious cholecystitis is a difficult if not impossible matter, if one endeavors at the same time to determine the nature of the infection and the character of the changes set up. Local tenderness, tumor in the region of the gall-bladder, jaundice, pain, and fever, are sufficient to establish a general diagnosis. The recognition of gangrenous or phlegmonous changes seems to resolve itself in the diagnosis of the causes giving rise to a septic peritonitis having its starting-point in the right upper abdominal quadrant. Unfortunately, there are several lesions capable of producing such a condition, as perforated gastric ulcer, appendicitis, perforation of the gall-bladder by calculous ulceration, and ulcer of the duodenum. Still, a mistaken diagnosis as related to these diseases is not a serious matter, for the treatment of each includes an exploratory laparotomy. It will be seen that the correct interpretation of the phenomena depends not so much upon present symptoms as upon an analysis of the patient's clinical history. Several times cases have been mistaken for acute intestinal obstruction, and operation for this performed.

3. Cancer of the Bile-Passages.

Cancer of the bile-passages is a very rare condition. In the majority of cases,—some claim invariably,—it has been preceded or accompanied by cholelithiasis. When the disease involves the gall-bladder, there is a distinct tumor formed in that locality. Its physical characteristics must vary according to the nature of the enlargement, *i.e.*, whether it is due in part to distention of the gall-bladder or to the cancerous growth. Van Lennep recommends as a good working rule, "That a distended gall-bladder with persistent jaundice means malignant disease; or, very rarely, stricture or cicatricial contraction; that a contracted gall-bladder with persistent jaundice means stone." Attendant symptoms are usually jaundice, pain and tenderness, and, towards the end, a general cachexia.

The diagnosis of cancer of the bile-ducts is even more uncertain than in the case of the disease involving the gall-bladder. It is rare that a palpable tumor is formed. Jaundice is, however, of more regular occurrence, and is persistent. The primary lesion is very liable to set up a suppurative cholangitis, in which case the diagnosis becomes more difficult. To quote Goodno, "Absence of a history of biliary colic, and an inability to discover any change in the liver by means of physical examination, suggests that a case of rapidly-developing and intense and persistent jaundice may be due to cancer of the bile-ducts, but there is always uncertainty as to the diagnosis."

4. Cholelithiasis.

In many instances,—it has been stated as ninety per cent.—the presence of calculi in the gall-bladder gives rise to no symptoms whatever. In the remaining ten per cent. the sufferings are at times agonizing, and it sometimes happens that the complications arising therefrom become a serious menace to life. When symptoms do arise, it is because the calculi have engaged in the biliary ducts or have produced one of the numerous complications. In the former case we have the condition known as *biliary colic*, the symptoms of which will present certain peculiarities according to the seat of the obstruction.

The characteristic symptoms of biliary colic include paroxysmal pain; vomiting, sometimes associated with collapse; fever; and, if careful search is made, the presence of calculi in the stools.

The pain is of agonizing severity. It comes on suddenly, and, after continuing for an indefinite period, it ceases as suddenly as it appeared. It is usually situated in the region of the gall-bladder, from which point it radiates into the epigastrium and the right shoulder. Keay declares—and herein his teachings differ from current opinions—that when the pain is dependent upon the engagement of a calculus in a duct, the starting-point of said pain is in the back, "somewhat to the right of the region between the eighth and the eleventh dorsal vertebræ." "This pain is felt only for a few minutes in its greatest intensity in that position. It gradually passes around, and is succeeded by pain in the right hypochondrium, the pain in the back disappearing, or being gradually merged in the more continuous pain over the gall-bladder. There, in the region of the gall-bladder, it holds its principal seat, but shoots about in all directions,—to the corresponding position on the left side, to the umbilicus or any part of the abdomen, but never in my (Keay's) opinion, to the right shoulder." Tenderness is nearly always present, not only during the paroxysms of pain, but during the interparoxysmal periods.

Vomiting is not always present. It may be paroxysmal or practically continuous. In some cases it may of itself constitute a source of danger, being productive of collapse. Profuse or frequent vomiting is, however, not to be regarded as a common symptom.

The presence of a tumor in the region of the gall-bladder is not often observed during the attacks of colic. When present, it is, according to Mayo Robson, due to the contraction of the muscular walls of the gall-bladder upon its contents. In the chronic cases it is frequently observed, and is then a valuable diagnostic symptom.

The most reliable symptom is the discovery of calculi in the stools. Inasmuch as their detection is by no means certain, their absence cannot be regarded in any other light than that of negative evidence generally. To make their discovery certain, the stools should be treated with a strong formalin solution as a deodorizer, after which they should be washed in a sieve having a mesh of $\frac{1}{16}$ inch.

So much, then, for the symptoms arising from gall-stones in general. These undergo important modifications according to the situation of the offending bodies, *i.e.*, as to whether they are in the cystic duct or in the ductus communis.

Obstruction of the Cystic Duct.—Jaundice is absent. The gall-bladder undergoes dilatation, and is readily palpated as a tumor which may at times attain quite a large size. The contents may consist of bile mixed with mucus, or, when the obstruction is chronic, clear fluid mucus. When the gall-bladder contains calculi and its walls are not very tense, palpation may elicit a distinct crepitus. As complications of obstruction of the cystic duct, cholecystitis of the simple, suppurative or phlegmonous variety, may set in, and add its symptoms to those of the primary trouble. Ultimately, there may be calcification or atrophy of the gall-bladder.

Obstruction of the Common Duct.—When the stone completely obstructs the duct, the most prominent symptom is the jaundice, which is well-marked and persistent. When the obstruction is incomplete, bile is observed in the fæces. In the case of complete obstruction, the stools are of an ashy color, and the urine contains bile. In incomplete obstruction, the jaundice is liable to considerable variations in intensity. In the case of a stone acting like a ball-valve, Osler has formulated as the special symptomatic features, (*a*) "Ague-like paroxysms, chills, fever, and sweat; the hepatic intermittent fever of Charcot; (*b*) jaundice of varying intensity, which persists for months, or even years, and deepens after each paroxysm; (*c*) at the time of the paroxysms, pains in the region of the liver, with gastric disturbance."

The above by no means exhaust the symptomatic possibilities of cholelithiasis, but they do present the principal features of uncomplicated cases. The presence of complications brings with it the special features belonging to each; it is therefore necessary only to mention them, and bear in mind the symptoms arising from each. These complications and sequelæ of gall-stones include the following: i. Ileus, due to atony of the bowel; ii. acute intestinal obstruction; iii. general hæmorrhages, the result of long-continued jaundice; iv. localized peritonitis producing adhesions, which may then become a source of trouble, even after the gall-stones have been discharged; v. dilatation of the stomach, depending upon adhesion around the pylorus; vi. ulceration of the bile-passages, establishing a fistula between them and the intestines; vii. stricture of the cystic or common bile-duct; viii. abscess of the liver; ix. localized peritoneal abscess; x. abscess in the abdominal walls; xi. fistula at the umbilicus or elsewhere in the abdominal walls; xii. empyema of the gall-bladder; xiii. suppurative cholangitis; xiv. septicæmia or pyæmia; xv. phlegmonous cholecystitis; xvi. gangrene of the gall-bladder; xvii. perforative peritonitis; xviii. extravasation of bile into the general peritoneal cavity; xix. pyelitis of the right side; xx. cancer of the gall-bladder or of the ducts; xxi. subphrenic abscess; xxii. empyema of the right pleura; xxiii. pneumonia of the lower lobe on the right side; xxiv. chronic invalidism and inability to perform any of the ordinary business or social duties of life. (Mayo Robson.)

CHAPTER VII.

THE SPLEEN.

Situation of the Spleen.—The spleen lies in the left hypochondrium immediately beneath the diaphragm. Its upper extremity is covered by the lower portion of the left lung, and hence is not definable by percussion. Its lower extremity extends to the eleventh rib. Anteriorly it reaches as far forwards as the mid-axillary line. Posteriorly it extends to within an inch and a half of the median line.

Methods of Examination.—The methods employed for examination of the spleen include:

1. Inspection.
2. Palpation.
3. Percussion.

1. **Inspection** is of very limited utility, as splenic enlargements, when sufficiently great to produce bulging in the left hypochondrium, are readily investigated by palpation.

2. **Palpation** likewise is of limited utility, as it can give information only when the spleen is sufficiently enlarged to extend beneath the costal margins. Ordinarily the recumbent position, with the shoulders raised, the thighs bent on the abdomen, and the abdominal muscles thoroughly relaxed, will give all available information. In doubtful cases, however, if the patient's condition is such as to permit it, the genu-pectoral position should also be tried. A very good position, also, is what might be called the diagonal position on the right side, that is, lying on the right side halfway between the dorsal and the right lateral position.

The normal position of the spleen is such that it cannot be felt by palpation. Exceptions to this are found in patients with decidedly relaxed abdominal walls, and those with deformed chest.

Palpation enables us to determine the mobility of the spleen. All splenic enlargements rise and fall with the movements of respiration, this fact ofttimes enabling us to determine the nature of a doubtful tumor. Exceptions to this statement are found in cases of enormous enlargements from leucocythæmia, in which, owing to the very great displacement of the diaphragm upwards, the respiratory movements of that muscle are prevented. Wandering spleen likewise does not move with the movements of respiration. In this case palpation recognizes the nature of the tumor by its shape, especially by its notches. The wandering spleen may assume positions far removed from the normal. Thus they have been found as low as the pelvic cavity. "Enlarged and wandering spleen lies in front of the colon." (Vierordt.)

Pain in splenic lesion, as in the case of the liver, arises from peritoneal irritation. In the case of enlargements, its intensity or its presence depends in great measure upon the rapidity with which the change has taken place. It is especially apt to be present in congestion attendant upon the acute infections; embolism, as in malignant endocarditis, tumors and abscesses. "There may even be tenderness of the ribs in the neighborhood of the spleen in cases of infarction and abscess."

3. **Percussion.**—Percussion, however, is the means affording the most information concerning the condition of the spleen, as by it the size of the organ, when it does not extend below the ribs, can be delimited accurately, even though it be normal or but slightly enlarged. It must be remembered, however, that that portion of the organ which is covered by the lung is inaccessible to percussion. The best results are secured by light strokes. The most favorable positions for the examination are standing, or with the patient lying somewhat on his right side. The area of splenic dullness is found to be rounded in front and below. Above, it is straight, as it is cut off short by the overlapping lung. Posteriorly the splenic dullness is not definable, because it merges into that of the left kidney and the lumbar muscles. Normally the area of splenic dullness should measure three-quarters by two inches.

The conditions of the spleen discoverable by physical examination include alterations in position, shape, size, and consistence.

(*a*) **Alterations in Position (Displacements).**—These may be either upward or downward. The latter are the more commonly observed in practice, and may result from

i. Rachitis.
ii. Left pleural effusion.
iii. Pulmonary emphysema.
iv. Relaxed ligaments (gravity).

Upward displacements can result only from pressure from below, as from ascites, tumors of the abdominal and pelvic viscera, and flatulent distention of the stomach and intestines.

(*b*) **Alterations in Shape.**—Most enlargements of the spleen preserve the same general contour as that presented by the normal organ, thus making the nature of the tumor readily recognized. Certain irregularities, however, occur in connection with several of the lesions. Thus in leucocythæmia the spleen is enlarged, and there are more or less numerous flat projections; in cancer, hard nodular tumors are observed. In this connection it is important to bear in mind that carcinoma of the spleen is practically impossible as a primary lesion. So, when this condition is suspected, it is important to determine positively that cancerous disease exists in other organs or tissues of the body. Small elastic or fluctuating tumors with a smooth surface indicate abscesses or cysts.

(*c*) **Alterations in Size.**—These include enlargements and atrophy. Enlargements of the spleen may occur in

i. Many of the infectious diseases, notably in typhoid and malarial fevers.
ii. In some of the chronic infectious diseases, as tuberculosis and syphilis.
iii. In the various anæmias, including leucocythæmia, in which the splenic enlargement may be truly enormous ; splenic anæmia, and Hodgkin's disease.
iv. Amyloid degeneration of the spleen.
v. Cirrhosis of the liver.
vi. Rachitis.
vii. Tumors, as malignant disease, and hydatid cysts.
viii. Abscess.
ix. Embolism by producing hæmorrhagic infarcts, especially in malignant endocarditis.

(*d*) **Alterations in Consistence.**—The soft enlargements of the spleen usually accompany the acute specific diseases, notably typhoid fever ; but the organ is rarely sufficiently large to be perceptible by palpation. The enlargements with increased consistence include amyloid degeneration, cancer, leucocythæmia, and malarial fever.

CHAPTER VIII.

THE PANCREAS.

Anatomy.—The pancreas is situated in the posterior portion of the epigastric and left hypochondriac regions. It is of oblong, tapering shape, and about six inches in length. Its right extremity is decidedly the thicker, and is known as the *head.* The left or tapering extremity is called the *tail.* The head of the pancreas fits in the concavity made by the curve of the duodenum. It is also in important anatomical relation with the portal vein, the superior mesenteric artery, the common bile-duct, the inferior vena cava, and the aorta. In front of the pancreas is the stomach. The tail of the pancreas is in relation with the lower extremity of the inner border of the spleen.

The secretion of the pancreas is discharged into the duodenum through a duct known as the pancreatic duct, or the duct of Wirsung, joining the common bile-duct in the ampulla of Vater.

Symptoms Resulting from Pancreatic Disease.—The clinical investigations of pancreatic disease have always been fraught with the greatest difficulty. Quite naturally, the pancreas being an organ of digestion, it is expected that disease of its structure should be attended by digestive disturbance. The number of organs concerned in this process being multiple, and it being the nature of each to compensate in a measure, at least, for the shortcomings of the others, it is almost impossible, in the absence of definite symptoms pointing unquestionably to the pancreas as the seat of disorder, to make a diagnosis of lesion of that organ. Physical examination affords us but little aid, because of the deep situation of the pancreas, which practically puts it out of reach of the methods at present available. One of the special functions of the pancreatic secretion is the digestion of fats. Hence for years it has been customary to regard the appearance of fats in the stools as evidence of pancreatic disease. But fatty stools may result from the too free ingestion of fatty foods, and they may be absent when the pancreas is diseased simply because other digestive fluids take care of the fatty elements of the food. In the absence, then, of positive signs special to pancreatic disease, one is forced to base his diagnosis upon careful analysis of the symptoms present, their possible relation not only to disease of the pancreas, but also to lesions of other organs, and the determination that such symptoms cannot be dependent upon any organic disease excepting of the pancreas. The problem is still further complicated by the knowledge that the pancreas is also an organ of internal secretion; but its functions in this respect thus far remain a matter of great mystery.

Subjective discomfort or actual pain is a common attendant upon pancreatic disease. Unfortunately, it cannot have much diagnostic value, because similar sensations may arise from disease of other structures underlying the epigastric region. Even when due to pancreatic disease, the subjective symptoms may arise from peritoneal involvement or interference with neighboring viscera, or they may be special to the particular pathological lesion present. When the subjective discomfort is due to pancreatic disease *per se*, it gives the impression of being deeply situated. If, under such circumstances, there is local tenderness, that tenderness is elicited by deep palpation. If, on the other hand, the pain is secondary to the pancreatic lesion, and is due to the effect of the same upon the overlying structures, it gives the appearance of having a superficial origin,—as if, for example, it originated in the stomach. Some cases of pancreatic disease are attended by severe agonizing pain, centreing in the epigastrium and radiating in all directions. Such pain is attributed by Roberts to implication of the solar plexus. It is often associated with restlessness, fainting, and even collapse.

The determination of disturbed pancreatic function is a difficult problem, for it is known that the pancreatic secretion is made up of no less than three ferments, to say nothing of the function of the gland as an organ of internal secretion. Such disturbance of function must involve excessive secretion, diminished secretion, and altered composition of secretion. Unfortunately, present clinical methods do not enable us to differentiate these in practice.

As evidence of increased pancreatic secretion, there was described in former years the so-called pancreatic diarrhœas, the stools of which consisted of a thin, viscid substance, and contained an abundance of leucin. It was admitted that this diarrhœa was the least constant of all the manifestations of pancreatic disease.

The symptoms arising from deficient pancreatic secretion must be the same, whether said deficiency is the result of defective quality or from some obstruction which prevents the pancreatic juice from entering the intestinal tract and there acting upon the food. These symptoms include those of disturbed digestion,—none of which, however, possess any diagnostic value,—the presence of fatty stools, and a diminution of indican in the urine. *Sialorrhœa*, or increased salivary flow, which was formerly regarded as an important sign of pancreatic disease, can only be present when there is associated lesion of the stomach.

The appearance of **fatty stools** has for ages been held as a most valuable sign of pancreatic disease, attention having been directed to it by Kuntzmann in 1820, and again by Elliotson in 1833. Of later years the reliability of this sign has been questioned, for it has been demonstrated that it is often absent in the presence of extensive pancreatic changes, and present when the pancreas is in nowise at fault. For example, in affections characterized by the absence of bile, as much as three-fourths of the fat taken with the food is passed out in the stools. A similar result may also ensue when the absorptive function of the intestinal tract is in abeyance. Inasmuch as the pancreas plays an important part in fat-digestion, it is reasonable to believe that some disturbance in its digestion must follow disease of that organ. Müller has shown that in the presence of pancreatic disease, there is no longer the normal degree of decomposition of fat in the intestinal tract. Normally, 84.3 per cent. of the fat in the intestinal contents is split up. From experiments, he argues that "a diminished decomposition of fat proves only that the flow of pancreatic juice is arrested, but does not in itself alone furnish any evidence that there is degeneration of the pancreas." To show the diminished decomposition of fat, Leo proposes the following procedure ("Twentieth Century Practice of Medicine," Vol. VIII, p. 377):

"The fæcal matter, dried at a temperature of 100° C. (212° F.), and finely pulverized, is treated for three days with ether in a Soxhlet apparatus. The ethereal extract is then evaporated to dryness, dissolved in absolute ether, filtered, dried, and, after being repeatedly washed with a small portion of warm water in order to remove the lower fatty acids and traces of soap, again dried, and weighed. This determines the amount of neutral fat and free fatty acids.

"A weighed portion of this mass is then dissolved in warm alcohol, with the addition of a sufficient quantity of ether, and then titrated with alcoholized caustic potash, with the addition of two drops of an alcoholic phenolphthalein solution as an indicator. This gives the free fatty acids.

"If the values so obtained are reckoned for the entire mass and subtracted from the previously-found total of fat and fatty acids, we obtain the amount of neutral fat.

"To determine the amount of saponified fat, the residue remaining after extraction by ether is boiled for a time in alcohol and hydrochloric acid, then dried, and again extracted with ether. After the evaporation of the ether, the residue is dissolved in alcohol with a little ether and titrated with caustic potash as above.

"As some of the soap is always removed with the ethereal extract, that is to say the fatty acids are withdrawn from a part of it, and as, furthermore, the relation of the fatty acids in the fæces to the soaps is an inconstant one and dependent on chance, we may, following Müller, refrain from an independent estimate of the soaps and free fatty acids. In this case, the dried and weighed portion of fæces to be examined may be at once treated with alcohol and hydrochloric acid, boiled for a time (whereby the soaps are converted into free fatty acids, but the neutral fats remain unaltered), and then extracted with ether. This extract is treated in the same way as the mixture of neutral fat and free fatty acids. The value so obtained for free fatty acids represents the sum of the previously existing free fatty acids and of those obtained from the soaps."

The presence of **clay-colored stools** in the absence of symptoms indicative of disease of the liver is regarded by Walker as symptomatic of pancreatic disease. He argued that the normal color of the fæces "depends upon the mutual reaction of the bile and pancreatic fluid under the influences met with in the intestinal tract." The absence of either of these secretions must therefore be followed by clay-colored stools. Which of these is at fault must be determined by the concomitant symptoms.

The pancreatic juice, in the exercise of its function, seems to have an important relation to the production of indican. It has been observed that the quantity of indican in the urine is much diminished when the pancreas is diseased. The importance of this sign is that of a differential one, for in digestive disturbances in which the stomach is at fault the quantity of urinary indican is increased.

When the clinical phenomena attendant upon disease of any viscus are as obscure as are those of the pancreas, one naturally seizes upon every suggestion offered, in the hope of throwing light upon an obscure case. Nencki proposes to utilize the effect of the pancreatic juice upon salol as a test. This drug is split up in the duodenum into salicylic acid and phenol, and is then eliminated in the urine. It may there be detected by the ferric chloride test.

Emaciation is present in many cases of pancreatic diseases. The origin of it, however, is obscure, for it does not seem to be dependent upon the absence of the pancreatic secretions, for it is present in cases in which the functional activity of the gland is but slightly impaired, and starches, fats and proteids are digested and absorbed.

The study of pancreatic diseases is made interesting as well as puzzling by the ability of the secretions of the organ to act upon the structure of the gland itself. This is undoubtedly in many instances a post-mortem change, especially when the pancreas is functionally active at the time of death. Extensive destruction of the pancreas from this cause has been observed even in cases in which the autopsy has been performed within three hours after death.

Fat necrosis is sometimes observed in connection with pancreatic lesions, especially in cases of acute and chronic inflammation and tumor of the organ, or in cases of obstruction of the pancreatic duct. It is believed that the condition "is due to the liberation of the fat-splitting ferment of the pancreas, which acts upon the cells of the pancreas and its neighborhood, converting the fat first into fatty acids, and next into salts formed from the combination of the fatty acids with calcium." Objectively, the lesions were manifested as areas of a dead-white color, generally small in size, and scattered through the pancreas and adjacent structures, even into the omental and parietal adipose.

Physical examination of the pancreas is unsatisfactory, owing to the deep position of the organ. It can only give positive information in cases of well-defined enlargement which are demonstrated to be situated behind the stomach.

Glycosuria.—It has long been known that certain cases of diabetes were found post-mortem to be associated with changes in the pancreas; hence a special variety known as *pancreatic diabetes* was recognized. But this information was of no practical value, because numerous cases of extensive pancreatic disease were observed, in which, during life, glycosuria was absent. These apparent discrepancies were finally explained by the researches of Opie and others, who have demonstrated, apparently beyond question, that disease of the islands of Langerhans is the essential lesion producing a pancreatic glycosuria. The whole subject is at present in a state of evolution, and much information of practical value may be shortly expected.

Synopsis of the Symptoms and Clinical Course of the Diseases of the Pancreas.

1. Pancreatitis.

Inflammation of the pancreas presents a variety of pathological changes. The acute parenchymatous variety is of unknown etiology, though it is believed to be the result of extension of disease from the duodenum. Its symptoms resemble very closely those of acute peritonitis,—so closely, indeed, that it is at times impossible to make a differentiation without an exploratory laparotomy. The symptoms include suddenly-appearing epigastric pain, radiating in various directions, associated with vomiting, fever, constipation, and tense abdominal walls. Within a comparatively short time the patient goes into collapse, and death ends the scene. Sometimes, however, the course of the disease is checked, or it merges into the chronic form; or suppuration may take place.

Suppurative pancreatitis is inclined to run a much more chronic course than the above. Aside from the constitutional symptoms of suppuration, the case presents icterus, fatty stools, glycosuria, and epigastric tumor, in typical cases. Osler reports a case correctly diagnosed during life by Thayer and successfully operated by Finney,—disseminated fat necrosis and deep-seated abscess with necrotic pancreatic tissue being found at the operation. Pancreatic abscess may rupture into the stomach, the duodenum, or the peritoneum, or it may lead to thrombosis of the portal vein.

Acute hæmorrhagic pancreatitis is of sudden onset, the first symptom consisting of severe epigastric pain, associated with nausea, vomiting and collapse. It will thus be seen that the clinical phenomena are far from characteristic. Fitz (quoted by Osler) gives the following data: "Acute pancreatitis is to be suspected when a previously healthy person or a sufferer from occasional attacks of indigestion is suddenly seized with a violent pain in the epigastrium, followed by vomiting and collapse, and in the course of twenty-four hours by a circumscribed epigastric swelling, tympanitic or resistant, with slight elevation of temperature. Circumscribed tenderness in the course of the pancreas and tender spots throughout the abdomen are valuable diagnostic signs."

2. Hæmorrhage into the Pancreas

May take place in the course of chronic interstitial pancreatitis, but under such circumstances possesses little clinical importance. In obese individuals with arterio-sclerosis there may take place free hæmorrhage, attended by severe epigastric pain, vomiting, collapse, and anxiety. The cases nearly always terminate fatally within a few hours. The severe collapse does not seem to be dependent upon the amount of blood lost. The diagnosis of these cases is practically impossible excepting on rare occasions, for the symptoms simulate very closely those of irritant poisoning, gastric or intestinal perforation, and cholelithiasis.

3. Chronic Interstitial Pancreatitis

Is nearly always secondary to disease of neighboring organs, as gastroduodenal catarrh or ulcerations, chronic peritonitis, tumors, biliary calculi, and aneurysms. Syphilis has been traced as a cause in a number of instances. The symptoms are not such as to enable one to make the diagnosis with any degree of certainty, especially as they are greatly obscured by the association with symptoms of the primary affection. The general phenomena indicative of indigestion,—as loss of appetite, nausea, flatulence, heaviness, and distention in the epigastrium,—constitute the first manifestations; after which may be noted emaciation and debility, disturbances of the intestinal function, colorless or fatty stools, and dull, boring pain. Glycosuria is sometimes present, and then affords a valuable diagnostic sign; but it is so frequently absent as to make it useless for diagnostic purposes.

4. Pancreatic Calculi

May be found at autopsy in patients who gave no evidence of their presence during life. As a rule, they are multiple. The symptoms they produce include epigastric pain, simulating in its paroxysmal character the pains of gall-stone colic. When, as sometimes happens, the calculus exerts pressure upon the common gall-duct, the symptoms must agree so closely with those of biliary calculus as to make a differentiation impossible. The paroxysmal pain, in association with the symptoms outlined as occasionally present in cases of pancreatic disease, sometimes leads us to correct conclusions.

Pancreatic calculi not infrequently lead to secondary changes, which include chronic interstitial pancreatitis, acute suppurative pancreatitis, and carcinoma from the prolonged irritation.

5. Pancreatic Cysts.

The majority of cases of pancreatic cyst are probably the result of obstruction of the duct of Wirsung or one of its branches. The lesions leading up to this result must be varied, including inflammation starting in the duodenum and travelling upwards, traumatism, and displacement of the pancreas. Traumatism is occasionally a cause, but an unusual one,—because, as Senn has suggested, the texture of the pancreas is so friable that severe blows produce extensive disintegration of structure.

Pancreatic cysts are remarkable, as a rule, for their chronic course, which may continue over many years. Their slow growth makes it possible for them to attain considerable size before there are any symptoms other than the enlargement. The situation of the latter depends in some measure upon the particular portion of the pancreas involved. When the tail is involved, the tumor is to the left of the median line. In the majority of cases, however, the tumor occupies the median line in the upper portion of the abdomen. It does not move with respiration. Percussion may show a band of tympany between it and the liver on the right, and the spleen on the left. On palpation, the tumor is globular and resistant. Posteriorly, dullness is increased on the left side, the line of the same approaching the angle of the scapula. Exploratory puncture removes a fluid of a reddish or dark-brown color, of alkaline reaction and specific gravity of 1010 to 1020, and containing pancreatic ferments, as shown by its ability to emulsify fats, convert starch into glucose, and digest fibrin and albumin.

When pain is present, it is situated in the epigastrium, from which it extends in various directions. There may also be associated gastro-enteric symptoms, including nausea, vomiting, and diarrhœa. The so-called orthodox symptoms of pancreatic disease, emaciation, fatty stools and glycosuria, are only exceptionally present.

CHAPTER IX.

THE NOSE.

In the clinical study of the nasal cavities, it is of the greatest importance that the anatomical structures involved and the functions of the part be kept in mind. This done, the examination becomes a comparatively easy matter. The nose is to be studied as

(a) The organ of smell ; and
(b) As a portion of the respiratory tract.

Anatomically, the nasal cavities are to be studied as consisting of a passageway for the transmission of air, and as consisting of mucous membrane, connective tissue, periosteum, cartilage, bone, and erectile tissue. In addition to these parts, the accessory sinuses, of which there are the frontal, ethmoidal and maxillary, must also be kept in mind as possible factors in the production of symptoms.

The External Appearance of the Nose.—Before proceeding with the study of the changes within the nasal cavities, a few words concerning the information to be derived from inspection of the nose externally will be advantageous.

The tip of the nose presents an abnormally red and swollen appearance in a variety of conditions, of which alcoholic excesses is the most important,—so important, indeed, that the mistake of overlooking other agencies applicable to the case in hand is too often committed. It is improbable that the red nose of alcoholics can exist without other visible evidence of the habits of the individual. Other conditions with which this symptom is associated are some cases of amenorrhœa, and gastric disorder.

Destruction or impairment of the bridge of the nose occurs in cases of hereditary and acquired syphilis, because of destructive disease of the bones and cartilages. Saddle-nose may also be an accompaniment of atrophic rhinitis, post-nasal adenoids, defective development, and traumatism.

The nostrils present a narrow or collapsed appearance in post-nasal adenoids, and in nasal obstruction from any cause. It is of interest to note that post-nasal adenoids are associated with a thickening of the tissues about the bridge of the nose, sometimes associated with enlargement of the transverse nasal vein, while the other extremity is narrowed.

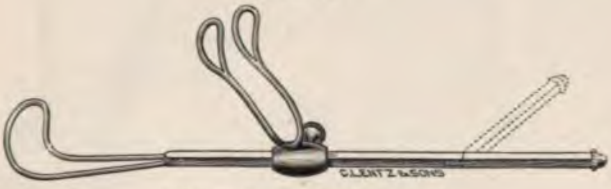
Dilatation of the nostrils with respiration occurs chiefly in children suffering from some disorder capable of causing difficult respiration. These include especially pneumonia, capillary bronchitis, laryngeal diphtheria, spasmodic asthma, and tetanus neonatorum.

The tip of the nose may become gangrenous as a result of frost-bite, Raynaud's disease, and syphilis.

Examination of the Nasal Cavities.

Anterior Rhinoscopy.—For the purpose of examining the nasal cavities, it is important that the parts be well lighted. Ordinary daylight does not give sufficient illumination, and sunlight is not always available. The best light will generally be found to be that of an argand burner mounted on a movable bracket, or a Rochester burner. It is always important that the observer's eye be in the line of illumination, hence a head-mirror is necessary. The light should be placed to one side and on a level with the patient's ear. The patient should be seated in a chair, the back of which is not too high to interfere with the free movements of his head. The physician should occupy an easy position facing the patient. To render the nasal cavities accessible to light and inspection a speculum is usually necessary. The kind used is very largely a matter of convenience on the part of the examiner. The bivalve specula, of which the well-known Kramer speculum is the type, are convenient, inasmuch as the operator can readily regulate the degree to which he separates the alæ nasi without causing discomfort. They are objectionable in some cases, as they are not self-retaining. The spring specula, of which, also, there are numerous varieties, have the advantage of being self-retaining, but are sometimes objectionable because they may, though rarely, cause pain.

FIG. 46.



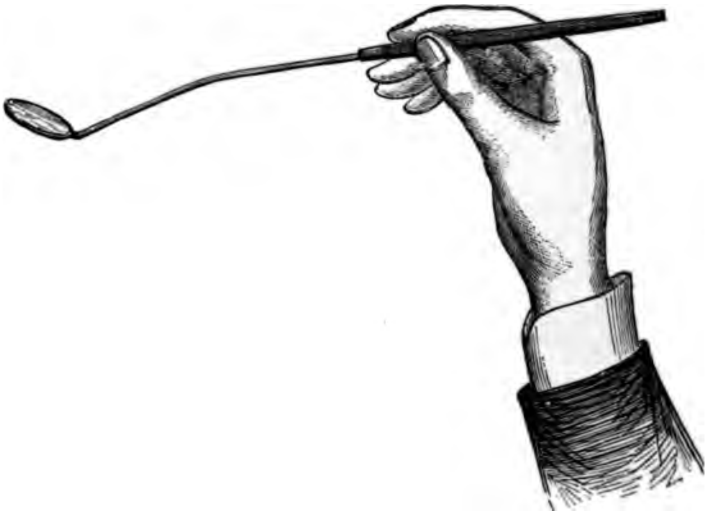
White's Soft-Palate Retractor.

The nasal cavities being illuminated, and the speculum being in place, attention should be directed to the following points: The patency of the passage, and, if obstructed, the nature and situation of the obstruction; the condition of the mucous membrane as to color, discharges and ulcerations; the conditions of the turbinated bodies as to size, color and ulcerations; the conditions of the septum as to shape, position, color and ulcerations; and the presence or absence of morbid growths.

Not all of a nasal cavity can be viewed at once. Anatomically, it is divided into an inferior, a middle and a superior meatus. The lower meatus is to be inspected with the patient's chin bent on the chest; the middle meatus is visible only when the patient holds his head forward or slightly upwards; while the roof of the nasal cavities does not come into view until the head is thrown well backwards.

Posterior Rhinoscopy.—Posterior rhinoscopy is rather an unsatisfactory method of examination excepting in the hands of the expert, mainly because of the necessity for attention to little details. The difficulties in practising it arise from the interference of the tongue and soft palate, the presence of enlarged tonsils, irritability of the throat, and lack of space posterior to the palate in which to place the rhinoscopic mirror. Skill in examination technique soon enables one to dispose of the tongue by means of a tongue-depressor; the soft palate may be gotten out of the way, if need be, by the use of a soft-palate retractor, of which White's is probably the best (Fig. 46). In some few cases in which a more efficient retractor is needed, elastic tapes may be introduced with Bellocq's canula, and fastened in front, thus bringing the soft palate well forward and out of the way of the field of examination. Irritability of the fauces may be overcome by painting the parts with a four per cent. solution of cocaine. The presence of enlarged tonsils is an insurmountable difficulty. Lack of room in the post-nasal space is rarely an important difficulty, because in most cases in which there is disease of the post-nasal space there will be found plenty of room for the placing of the mirror.

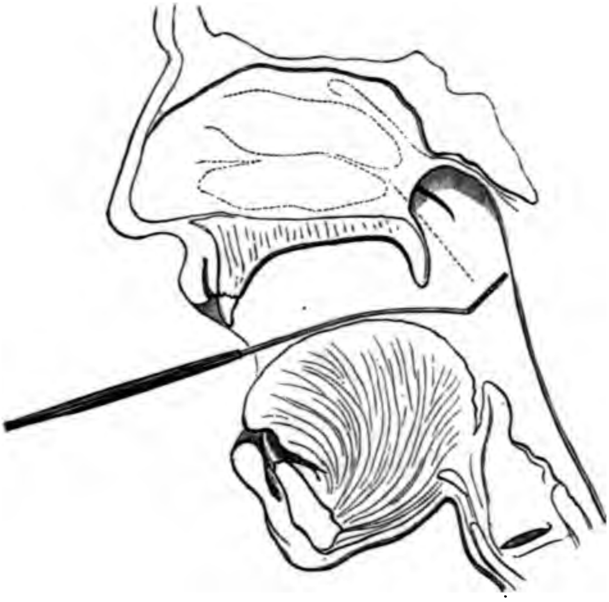
FIG. 47.



Curve of shank of mirror and position of hand necessary for rhinoscopy.

When making the examination, the patient's head should be made to incline slightly forward. The smallest-sized throat-mirror, with the shank bent as in Fig. 47, to conform with the curve of the floor of the mouth, should be introduced into the pharynx, care being observed not to touch any of the parts in so doing. In some cases this may be impossible. The tongue should be controlled by gentle but firm pressure with a tongue-depressor. If the uvula tends to get in the way or to contract, the patient should be directed to breathe slowly through the nose. Coming next to the inspection of the parts, it must be remembered that it is utterly impossible to view more than a small portion of the post-nasal space in the mirror at any one time. It is necessary, therefore, to shift the mirror from time to time, and thus obtain a conception of the various parts, from which a complete picture of the post-nasal space may be constructed. The accompanying figure gives a correct idea of the relations of the parts in this locality.

FIG. 48.



Section showing position of mirror and patient's head for obtaining a rhinoscopic image.—After Lennox Brown.

The lesions to be observed include, for the most part, adenoid and cicatricial bands and adhesions, and alterations about the openings of the Eustachian tubes.

In children, posterior rhinoscopy is always unsatisfactory, and it is in them that we most frequently find a careful examination of the post-nasal space necessary. It is then advisable to supplement the use of the mirror with digital examination of the parts. Normally, the post-nasal space is perfectly smooth, with the exception of the cartilaginous tips of the Eustachian tubes and the internal pterygoid processes.

Normally, **the mucous membrane** of the nose presents a pinkish color, which is characteristic. It is *abnormally pale* in anæmia from any cause, as are also all other mucous membranes. Its *redness is increased* in all cases of congestion and inflammation, and it presents a peculiar *whitish redness* in cases of atrophy.

The inflammatory affections giving rise to increased redness include

a. Acute catarrhal rhinitis.
b. Chronic catarrhal rhinitis of all varieties.
c. Hay-fever.
d. Certain infectious diseases, notably measles, scarlatina, typhoid fever, and diphtheria.
e. Chronic infections, including syphilis, tuberculosis, scrofulosis, etc.
f. Local irritation, as from foreign bodies, tumors, etc.

These affections are to be differentiated not by the color of the mucous membrane, but by associated conditions. In some the history of the case, and in others the associated objective conditions, as the discharge and the presence or absence of ulceration, and turbinated enlargement, decide. It should be remembered that obstruction of the nasal cavities, preventing the free ingress and egress of air, constitutes a most important etiological factor in bringing about a changed appearance of the nasal mucous membrane.

The state of the mucous membrane as to thickness should be studied. It is thickened in hypertrophic catarrh and most inflammatory affections, while it is unduly thin in atrophic catarrh. The latter condition is usually associated with atrophy of the turbinated bodies. A distinction should be attempted between a swelling due to inflammation and an actual thickening owing to a hyperplasia of connective tissue. The former is the condition observed in acute catarrhal rhinitis, while the latter is found in chronic catarrhal inflammations. In some few cases, the mucous membrane is œdematous. This condition occurs in cardiac and renal affections and chronic inflammations.

The mucous membrane may present a relaxed appearance, this being the case in old catarrhal inflammations in persons of generally depreciated constitution.

Ulcerations of the nasal mucous membrane may occur as follows: (a) Simple; (b) syphilitic; (c) tuberculous; (d) lupoid; and (e) leprous.

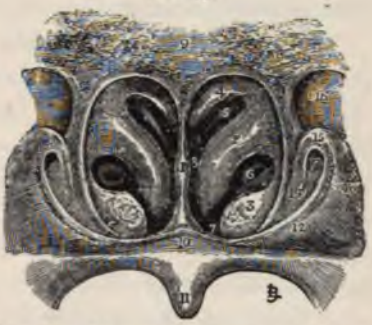
The simple ulcers include (i) catarrhal; (ii) herpetic; (iii) eczematous; (iv) traumatic; (v) neuro-paralytic; (vi) scorbutic; (vii) diabetic; (viii) varicose.

Simple catarrhal ulcerations are always superficial. They seem to be due to the irritation of the abundant discharge, and are especially liable to occur over swellings and prominences where the secretions may lodge. They are sometimes painful; but they are of short duration.

Herpetic Ulcerations occur at the margins of the nostrils, and succeed typical herpetic vesicles. They are common accompaniments of acute rhinitis, and are analogous to herpes of the lips.

Eczematous ulcers are observed especially in children. They are associated with eczema of the face or of the skin about the nostrils, and with cracks or fissures about the alæ nasi. Crusts are commonly present, and itching is an important symptom, as the scratching and picking excited by it are important producing factors.

FIG. 49.



Posterior rhinoscopic image: 1. Septum. 2. Middle turbinated bone. 3. Inferior turbinated bone. 4. Superior turbinated bone. 5. Superior meatus. 6. Middle meatus. 7. Inferior meatus. 8. Main passage of meatus. 9. Vault of pharynx and pharyngeal tonsils. 10. Cushion of soft palati. 11. Posterior surface of mouth. 12. Ridge formed by levator palati. 13. Salpingo-pharyngeal fold. 14. Salpingo-palatal fold. 15. Eustachian prominence or cushion. 16. Fossa of Rosenmüller. 17. Eustachian orifice.

Traumatic ulcers are due to picking of the nose or to the presence of foreign bodies. They disappear promptly on the removal of the offending substance.

Neuro-paralytic ulcers are very uncommon. They are due to paralysis of the fifth pair of cranial nerves. They are characterized by their painless, sluggish course, and the absence of discharge. They show no tendency to heal; but they do not extend to the skin. They may perforate.

Scorbutic ulcers nearly always occur in association with other well-marked symptoms of scurvy, and are generally due to some accidental local irritation in the course of that disease. They are attended by fœtor; the edges of the ulcerations are hard and thick, and the surface is fungoid and bleeding.

Diabetic ulcerations are dependent upon the low grade of nutrition of that disease plus the presence of some local irritation. They are especially liable to occur near the nasal orifice, as the result of scratching or picking.

Varicose ulcerations are associated with venous congestion of the mucous membrane. They are exceedingly sluggish, and bleed easily. They are bluish-red in color, irregular in outline, and superficial.

Syphilitic ulcerations may occur in either the primary, secondary or tertiary stages of the disease. The *primary syphilitic ulcer* of the nose is the chancre. It is very rarely observed, and presents the features of the chancre occurring in other situations.

The superficial ulcerations of secondary syphilis and the mucous patches present no features to distinguish them from similar lesions in other situations, *e.g.*, the mouth. Their nature is to be determined by the history of the case and their association with other mucous membrane manifestations of syphilis.

The **ulcerations of tertiary and hereditary syphilis** are really the most important. They may occur in any portion of the nasal cavities, and are often exceedingly destructive. Starting as excoriations, they may become deep serpiginous ulcerations, extending to the bones and cartilages, destroying them, and producing perforations or necrosis and deformities. The ulcerated surfaces are covered by a dry, greenish crust.

Tubercular ulcerations are usually found on the septum. They present a whitish-gray surface, with irregular and poorly-defined edges. They bleed very readily. The surrounding mucous membrane may exhibit small miliary tubercles. Microscopic examination of the scrapings will discover the presence of tubercle bacilli.

Lupoid ulcers are regarded by many as identical with tuberculosis. Their distinctive clinical feature, however, is that they have their starting-point on the skin, from whence they extend into the nasal cavities.

Leprous ulcerations are also the result of extension from without. They start with the leprous nodules of the skin. These undergo ulceration, with offensive, watery discharge. The diagnosis is made, however, by the character of the associated skin-lesions. They are very destructive, especially of the cartilaginous septum and the alæ nasi.

The ulcerations of malignant disease are always secondary to the formation of a tumor. Hence they are best studied in conjunction with the new growths of the nasal cavities.

Post-febrile ulcerations occur after measles, scarlatina, typhoid fever and variola. They are usually situated anteriorly on the septum or just inside the alæ nasi, and are generally very irritable. The history of the case decides the diagnosis.

The **discharges** in the nasal cavities may consist of

a. Mucus, which is often so thin as to merit the term serous.
b. Muco-purulent.
c. Purulent.
d. Crusts.
e. Blood.
f. Caseous materials.
g. Membrane.
h. Cerebro-spinal fluid.
i. Rhinoliths.

The microscopical and chemical character of nasal discharges is not without interest, although not often of value, clinically, in deciding as to the nature of a case under investigation. The normal nasal mucus is small in quantity when the vascularity of the secreting structure is considered. It is found to contain squamous and ciliated epithelium, leucocytes, and numerous fungi. It has an alkaline reaction, and contains considerable mucin. These epithelial cells exist in greater abundance in acute catarrhal rhinitis. The bacilli of tuberculosis and leprosy may be discovered in the discharges of the ulcerations from their respective lesions. In cases of ozæna the micro-organisms present themselves in great variety, bearing no relation whatever to the special objective and subjective features. In a few cases the thrush fungus has been found, and the Charcot-Leyden crystals have been discovered in the nasal secretions of asthmatic subjects and in connection with polypi.

The characteristic **mucous discharge** from the nose consists of a thin, watery fluid, which may or may not excoriate the parts over which it flows. It is an important phenomenon of the early stages of acute catarrhal rhinitis, hay-fever, and nasal hydrorrhœa.

In **acute catarrhal rhinitis** the discharge is at first scanty, becoming more profuse with the progress of the disease. Its superabundant saline constituents cause it to excoriate the skin over which it flows. It is apt to accumulate in the passage which happens to be the lower when the patient reclines, although this condition occurs in any case in which the discharge is both profuse and fluent, as it depends upon quantity and gravity entirely.

In **nasal hydrorrhœa** the discharge is watery and profuse. It is unattended by inflammation. The flow comes on in paroxysms at variable intervals, and continues for a longer or shorter time, and then disappears as mysteriously as it came. The chief annoyance arises from the profuseness of the discharge. This trouble seems to be of neurotic origin. It may result from the irritation of nasal spurs or obstructions. In a case seen by the author, it disappeared permanently after cauterization of the turbinated bodies. During the paroxysms, the mucous membrane becomes swollen and congested.

Hay-fever is characterized by its seasonal recurrence and the long continuance of the attack. Excepting for the severity of the symptoms, the clinical picture is that of the acute stage of catarrhal rhinitis. In persons predisposed to the affection, the initial symptoms appear on almost precisely the same day each year, unless it should happen that the victims are residing for the time being in a portion of the country in which hay-fever is unknown. The coryza is usually profuse. It is watery at first; later, it becomes purulent. The associated swelling of the mucous membrane of the nose causes great suffering. Sneezing is a more or less constant source of annoyance. In many instances chest-symptoms appear and complicate the case. These include bronchitis of more or less severity, and paroxysms of asthma. Attempts have been made to associate hay-fever with local changes within the nasal cavities, but thus far without any practical result.

Coryza constitutes one of the important symptoms of **infantile syphilis.** Indeed, it may be the initial symptom, so far as the knowledge of the parents is concerned. In the beginning, the discharge is watery (mucous), and is interpreted as an ordinary acute rhinitis. But it is very persistent, and changes to purulent or sanguino-purulent character. It dries and forms crusts obstructing the nares. The portions of the lips with which it comes in contact are excoriated. The nasal mucous membrane undergoes thickening, and not infrequently ulceration. Finally the discharge becomes offensive. Disease of the bones and cartilages, with nasal deformities, are common terminal results.

(*b*) A **muco-purulent discharge** is seen in the second stage of acute catarrhal rhinitis, chronic catarrhal rhinitis and chronic hypertrophic rhinitis. Occurring during the course of acute catarrhal rhinitis, it succeeds the watery discharge above referred to. It presents itself as a thick yellowish or yellowish-green discharge, usually non-irritating, but sometimes excoriating the portions of the skin adjacent to the nostrils. It is often profuse, and in some persons may continue for a long time after the active symptoms of the acute cold have disappeared.

There is nothing distinctive of the muco-purulent discharge of the chronic varieties of rhinitis. Whether the fundamental disorder is a simple catarrhal, a hypertrophic, or a scrofulous rhinitis, must be determined entirely by the associated symptoms.

(*c*) **Bloody Discharge.**—The discharge of blood is generally known as epistaxis. Like all hæmorrhages, it must depend upon some disturbance in the integrity of the parts, for without such a lesion the blood could not escape. At the same time, its existence is often favored by vascular degeneration, alterations in blood-pressure, and changes in the composition of that fluid. Careful inspection will nearly always enable the physician to discover the point from which the blood escapes.

The conditions which may produce epistaxis are traumatism, *e.g.*, picking at the nose, and blows ; typhoid fever, and some other acute infectious diseases ; anæmias, especially chlorosis and pernicious anæmia ; hæmophilia, plethora, puberty, local ulcerations, and amenorrhœa.

The epistaxis of the early stages of *typhoid fever* is well-known. Indeed, some authorities regard its presence as essential to the early diagnosis of that affection. It seems to bear a close relation to the condition of the blood and alterations in blood-pressure. Other fevers in which it may occur include malarial fevers, typhus fever and scarlatina.

The epistaxis of *chlorotic young women* is rarely sufficient to occasion alarm. This variety seems to be a diagnostic stumbling-block, for the frequent bleeding may be accepted as the cause and not the result of the anæmia. Then, too, owing to the amenorrhœa accompanying chlorosis, the epistaxis may be looked upon as a vicarious menstruation. The history of the case and an examination of the blood should prevent diagnostic error. Pernicious anæmia and leucocythæmia, with their hæmorrhages from the nose and other parts, may likewise escape proper recognition. In the early stages of the disease, it is hardly likely that the physician will resort to an examination of the blood, although the persistent recurrence of the bleeding and the association with hæmorrhages in other parts of the body should be suggestive.

In **hæmophilia,** the condition of the blood is such as to favor bleeding from slight causes. The patient will give a history of persistent hæmorrhage following tooth-extraction and other slight surgical injuries. The non-coagulability of the blood will be self-evident.

While epistaxis may occur in **scurvy,** it is hardly likely to be an initial symptom. The associated sponginess of the gums and the diet of the patient establish the diagnosis.

Changes in vascular pressure constitute an important cause of many cases. This is the case in the epistaxis of **plethora,** in which the bleeding gives relief to the full feeling of the head. It is doubtless, too, the proper explanation of many of the cases of so-called vicarious menstruation. The amenorrhœa leads to increased vascular pressure, with rupture of the delicate blood-vessels of the nasal mucous membrane.

In people of advanced or advancing years, epistaxis is often associated with **chronic interstitial nephritis,** being dependent upon vascular degeneration and increased arterial tension.

The epistaxis of **puberty** is probably the result of circulatory irregularities accompanying the developmental period of life.

As a rule, the loss of blood in epistaxis is unimportant; sometimes, however, it becomes so profuse and continuous as to endanger life. In all such cases it is of the greatest importance that the nasal cavities be examined carefully for the *locus mali,* for its discovery is, as a rule, sufficient to suggest measures for its prompt suppression.

(*d*) **Purulent Discharge.**—A purulent discharge can occur only in the presence of suppuration. Hence it indicates either abscess or ulceration. The latter as a cause is evident on inspection. Abscess in this locality is practically always due to disease of the accessory sinuses; in which case, also, it is noteworthy that the discharge is unilateral. In this connection it may be observed that the other important cause of unilateral nasal discharge is the presence of a foreign body, in which case the discharge may be muco-purulent, purulent or bloody, according to the character of the resulting ulcerations.

Improvements in examination technique of late years have demonstrated the frequency with which nasal discharges are dependent upon disease of the accessory nasal sinuses,—a frequency that passes the comprehension of all excepting the workers in this special field. For this reason, it seems to the writer that a review of the subject is not amiss, notwithstanding the lesions are strictly of a surgical character so far as treatment is concerned. The accessory nasal sinuses include four on each side, namely: i. The antrum of Highmore; ii. The ethmoidal sinus; iii. The frontal sinus; iv. The sphenoidal sinus.

The explanation for the frequent occurrence of sinus disease is found in the fact that the mucous membrane lining these cavities is an extension of that lining the nasal fossæ; hence disease in the latter location may, under favorable circumstances, spread to the sinuses. In the ordinary "colds," the danger of such extension is not of much importance. But many of the acute infectious diseases are attended by changes in the mucous membrane of the nose, and it not infrequently happens that such changes form the foundation for sinus suppuration. Especially does this remark apply to influenza, although other diseases, as erysipelas, measles, typhoid fever, scarlet fever and diphtheria, may prove to be important etiological factors. In the case of the antrum, the irritation and subsequent infection favored by a carious tooth is, beyond all question, the most important cause.

The very free communication between the various sinuses makes the simultaneous involvement of several of these cavities a matter of relatively common occurrence.

Disease of the Antrum of Highmore or Maxillary Sinus.—The symptom which drives the patient to the physician for relief is a purulent discharge, and this to the sufferer has an unpleasant odor, which is not perceptible to others. This symptom is important in that it is the direct antithesis of what is observed in atrophic rhinitis, in which disease the discharge is offensive to bystanders, but not to the patient. Exceptionally, the discharge in antral disease may be offensive. It is usually yellowish in color; sometimes it is of a brownish-yellow hue; or, again, it may be green and viscous. The nasal passage is not infrequently obstructed, owing to the presence of polypi or inflammatory thickenings of the mucous membrane in the middle meatal region. Associated symptoms, which, however, do not possess a diagnostic value, but nevertheless are of importance as adding greatly to the patient's sufferings, include headache, neuralgia, indigestion, mental lassitude, and a feeling of general ill-health. In the acute inflammation of the antrum, with pent-up discharges, there is intense pain in the cheek, with redness and possibly œdema of the overlying parts. The influence of the position of the head upon the discharge is one of the most important diagnostic symptoms. Before observing on this point, it is necessary that the middle meatus be carefully cleansed with a cotton-tipped probe. Then the patient is instructed to recline his head with the suspected side uppermost for about five minutes. If there is pus in the antrum, the discharge will reappear in the middle meatus at the end of this time. Valuable as is this sign, it may be absent. The patient may have emptied the sinus before the consultation; or the discharge may be too thick to escape in this manner; or there may have been temporary obstruction of the orifice of exit. The appearance of mucous membrane swellings, granulation tissue, and polypi, must always be looked upon as of especial diagnostic value when found in the middle meatus. Transillumination of the antrum by a specially-constructed electric lamp placed in the mouth is a valuable, though not infallible, means of diagnosis. The thickening of the mucous lining of the antrum interferes with the transmission of light through the cheek on the affected side. It sometimes, though rarely, happens that the bony walls of the maxillary sinuses are not of equal thickness on the two sides, in which case transillumination fails to possess any diagnostic value. The diagnosis of maxillary sinus suppuration can never be assured until an exploratory puncture has been made and the cavity irrigated. This operation is one entirely devoid of danger, even when performed unnecessarily. It is always to be preferred to the destruction of a useful tooth.

Disease of the Ethmoidal Sinuses.—Empyema of the ethmoidal sinuses may be closed or open. In the former cases, which are rare, the patient complains of more or less severe aching over the bridge of the nose, and nasal obstruction. Objectively, the middle turbinated is greatly distended, and manipulation with a probe discovers an "egg-shell crackling."

In open empyema of the ethmoidal sinuses there is purulent discharge from the nose, in addition to the pain over the bridge. The severity of this pain varies according to the freedom with which the purulent accumulation can escape. When the disease has continued for a long time, the tissues of the upper portion of the nose undergo extensive thickening.

An objective examination reveals the presence of polypi, and, after their removal, granulation tissue in an abnormally capacious middle meatus. Manipulation with the probe demonstrates in many instances the presence of caries and necrosis of the ethmoidal cells.

Disease of the Frontal Sinus.—The symptoms of acute suppuration within the frontal sinuses are supra-orbital pain, tenderness on pressure over the lower part of the forehead, sometimes œdema and swelling of the upper eyelid, and fever. When the outlet of the sinus is closed, thus confining the secretions under pressure, the pain may be intense. In mild cases, the only symptom may be the supra-orbital pain, which is too frequently mistakenly diagnosed as neuralgia. The diagnosis in doubtful cases is based upon the history of the illness. Most cases originate in nasal catarrh secondary to acute infectious diseases, especially to influenza.

The symptoms of chronic suppuration within the frontal sinuses include frontal headache, the discharge of pus, and the formation of polypi and granulation tissue within the nasal cavity. The severity of the headache is dependent entirely upon the tension under which pus is confined within the affected sinus. The presence of polypi and granulations in the middle meatus possess the same clinical importance as in the case of suppuration within other sinuses.

The principal diagnostic difficulty encountered is the determination of the presence of suppuration within other sinuses, especially the maxillary and the ethmoid. The existence of these must be confirmed or refuted by examination along the lines already laid down when describing the clinical features of these lesions.

Disease of the Sphenoidal Sinus.—The characteristic symptoms of suppuration within the sphenoidal sinuses include severe pain in association with ocular symptoms and post-nasal purulent discharge. Unfortunately, the frequent association of suppuration within other sinuses obscures the symptomatology, in which case the associated lesions must first be cleared up by treatment. The ocular symptoms include retro-bulbar neuritis, ocular pain and tenderness, and rapid blurring of vision, especially at the periphery of the field, and slight exophthalmos. The discharge of pus is usually not great. It tends to accumulate between the middle turbinated and the septum, or it may be seen posteriorly by the rhinoscopic mirror at the posterior termini of the middle and inferior turbinated bodies.

In doubtful cases, probing of the sinus, as in the case of disease within the other accessory nasal cavities, should be tried.

(*e*) **Crusts.**—These are the result of evaporation of the watery constituent of the nasal secretion, leaving the desiccated epithelium and saline materials behind. They occur more or less in health, although never to the extent of being an annoyance. Pathologically they may be found in any local condition which causes a retention of secretion and undue entrance of air to promote evaporation. Hence they are especially apt to occur in atrophic nasal catarrh, the patency of the nasal canal favoring both retention and evaporation. Sometimes they undergo decomposition and give rise to an offensive odor.

Crusts may also originate in the scabbing over of ulcerations with their discharge.

(*f*) **Caseous materials** accumulate only in that very rarely observed condition, caseous rhinitis. They present a cheesy gelatinous appearance, and seem to bring about disease of the bones and cartilages and facial deformity. The material is readily broken up with a probe, and the subjacent mucous membrane is found to be of a dull red color.

(*g*) **Membrane.**—The disease *par excellence* attended by membranous formation in the nasal passages is diphtheria. It very rarely, however, exists in this locality as the sole expression of disease, being nearly always attended by pharyngeal diphtheria, of which it is a direct extension.

A **non-diphtheritic membranous rhinitis** is claimed by some authors. It differs from the diphtheritic variety in that it is a purely local disorder, and in the character of the membrane, which is brighter in color and pearly in tint as compared with a dirty gray or white in diphtheria. It is, moreover, superficial, while the diphtheritic membrane is closely adherent, and involves the deeper layers of the mucous membrane. The simple membranous variety rarely leaves a bleeding surface on removal, and its discharge is not offensive.

(*h*) **Cerebro-Spinal Fluid.**—The discharge of cerebro-spinal fluid from the nose is rarely observed, as it occurs only in connection with certain perforating wounds of the cranium, and intracranial tumors. It is readily recognized by chemical analysis, as it contains no albumin, and it exerts a reducing action on copper solutions, as if it contained grape-sugar.

(*i*) **Rhinoliths** are rarely observed. They give rise to about the same symptoms as foreign bodies.

The Turbinated Bodies.—The turbinated bodies constitute an important objective point in every nasal examination. Their color may be altered in the direction of unusual pallor or increased redness ; or they may be enlarged by reason of vascular turgescence or actual hypertrophy, or diminished in size because of atrophic changes.

Pallor is observed in conjunction with a similar condition of the balance of the nasal mucous membranes. They are also abnormally light when they have undergone atrophic changes. It is unusual for them to present **undue redness** without having at the same time undergone increase in size. The latter condition is observed in all inflammatory affections of the nasal passages excepting those of atrophic character. In many of these cases, as in acute catarrhal rhinitis and the more recent cases of chronic catarrhal rhinitis, the enlargement is dependent upon vascular turgescence. This may be demonstrated by the application of a 4 per cent. cocaine solution, which produces prompt reduction of size. In cases in which there is hyperplasia of connective tissue, cocaine exerts little or no effect.

Hypertrophy of the turbinated bodies occurs in nasal catarrhs of long standing, the engorgement finally promoting connective tissue formation. It is thus that chronic hypertrophic rhinitis originates. Investigations have demonstrated that the changes are extensive. The mucous membrane is thickened throughout. The glandular elements are replaced to a certain extent by connective tissue, and there is increase of the latter in the submucous layer. Sometimes myxomatous changes are present, these being especially noticeable about the inferior and anterior end of the middle turbinated, giving rise to the impression that a polypus is present.

The extent to which the hypertrophies may encroach upon the nasal passages is variable in different cases. In extreme examples the hypertrophied turbinateds reach all the way across and impinge on the septum. Sometimes, too, the greatly-thickened mucous membrane hangs in folds, thus occluding the inferior meatus. In some of the latter cases it may simulate a polypoid growth very closely.

The thickening of the nasal mucous membrane is readily discovered by manipulation with a probe, which causes a pit on pressure, disappearing very slowly.

No subject concerning nasal pathology has been more discussed, with less result, than that of **intra-nasal atrophies,** or **atrophic rhinitis,** as it is commonly called. However well all observers may agree concerning the objective appearances, the widest possible diversities of opinion exist as to their modes of origin and the explanations for the existence of certain symptoms. In view of this fact, the object of this work will be better served if all such theories are neglected and our study be confined to the physical condition of the nasal cavities in this most intractable disease.

A remarkable roominess of the nasal cavities is the first thing that strikes the examiner. This may be so extreme as to make an inspection of the posterior wall of the pharynx possible through the anterior nares. The turbinated bodies are small, and the nasal cavities are lined more or less completely with crusts, beneath which is an offensive pus, or muco-pus. These are exceedingly difficult of detachment, and, when removed, the underlying mucous membrane will probably be found to be unduly red, owing to the mechanical interference. This redness subsides shortly, leaving it abnormally pale and thin. Bleeding takes place in response to very slight injury. Sometimes the mucous membrane looks shrunken and somewhat glazed. The mucous membrane of the pharynx participates in the changes observed in the nose, it also being pale, thin, dry and glazed. There is a strong disposition on the part of some rhinologists to regard atrophic rhinitis as a local expression of a constitutional state, as syphilis, tuberculosis, or scrofula; and, while this may be the case in many instances, it is by no means of universal application, for many of the sufferers are in most excellent health, other than the local disturbance.

The Septum.—It is generally stated that no person has a perfectly straight nose, the tip deviating to the right in right-handed persons and to the left in the left-handed. These deflections are in all probability due to the manner of wiping the nose, thus moulding that organ into its abnormal shape. The septum nasi, likewise, must not be expected to be perfectly straight, dividing the nasal passages into cavities of equal size. These remarks seem to the writer to be of especial importance, because there is a large school of rhinologists eagerly bent on the correction of every septal deformity, however slight, as the *fons et origo* of every discomfort to which the nose is heir. This fad brings to mind the division of gynæcologists, twenty years ago, into two classes,—one engaged in making cervical lacerations, the other in sewing them up.

Slight irregularities of its surface are very common. On its upper portion is a slight bulging, known as the tubercle, formed of an aggregation of glandular structure in the thick mucous membrane. This must not be mistaken for a deflection.

These normal or comparatively normal conditions aside, the septum may present conditions of the greatest clinical importance, as they may give rise to widespread changes in the nasal fossæ. These changes include deflections (congenital and acquired), spurs, tumors and perforations.

Whenever inspection shows a protrusion of the septum into one nasal cavity, examination of the other side should be made at once to determine whether there is a corresponding depression, as in that case the condition is one of deflection. If no concavity is found, then there must be a thickening.

The causes leading up to nasal deflections and thickenings are too numerous to be mentioned here. Indeed, to enumerate them would hardly subserve the purposes of a clinical work like the present. The fact that they exist and require correction is all-important. Their presence is harmful in that they obstruct nasal respiration, and, if they come in contact with neighboring structures, tend to accumulation of secretions and local irritations.

Perforations of the nasal septum are in the majority of cases of syphilitic origin. Exceptions are sufficiently numerous, however, to make this symptom of but little value in diagnosing the existence of syphilis, as it may occur from the irritation of a dusty atmosphere, chemical fumes, traumatism, as constant picking of the nose, malignant disease, and from ulcerations attendant upon the specific infectious fevers.

Acute abscess of the septum as a cause of thickening should be readily diagnosed, especially so in view of its acute onset. As is the case with all suppurations, there must have been opportunity for infection, and this is afforded in the cases under consideration by traumatism, infectious fevers,—notably measles, scarlet fever and typhoid fever,—erysipelas, uric acid diathesis, and tuberculosis. The fluctuating character of the swelling common to all abscesses is present. Localized at first, the pus tends to spread beneath between the perichondrium and the septal cartilage, extending even to the tip of the nose. The condition will, of course, be associated with all the usual concomitants of acute suppuration, as pain, extensive swelling of adjacent structures, and sometimes suppurative fever. If the pus is not evacuated promptly, destruction of cartilage and deformity of nose may ensue.

Chronic abscess is not attended by the severe subjective phenomena of the acute variety. Indeed, the fluctuating swelling and the nasal obstruction to which it gives rise may be the only symptoms. It usually involves the anterior part of the cartilaginous septum, and is secondary to cartilaginous disease resulting from typhoid fever, syphilis, or tuberculosis.

The only other fluctuating swelling over the septum is *hæmatoma*, which is due to the sudden effusion of blood into the submucous tissues, usually the result of traumatism. The history of the case should prevent diagnostic errors.

Nasal Tumors.—Many varieties of tumor may attack the nasal passages, but those known as *polypi*, and consisting of *myxomatous* tissue, are so much more frequent than the others as to have mainly engrossed professional attention. They present themselves as semi-transparent masses, sometimes single, but more frequently multiple, and usually attached by a pedicle. They vary in size from the smallest possible growth to one filling the entire nasal cavity, even to the extent of protruding from the nostril. They arise most frequently from the middle turbinated body or adjacent structures. The symptoms produced by them are mainly mechanical, *i.e.*, obstruction of respiration and its attendant reflex phenomena.

Fibromata in the nasal cavities are noteworthy, mainly because of the profuse hæmorrhages to which they give rise, and the attendant stenosis and deformities. The pressure they exert shows itself externally, causing deformities of the face, and in some cases they have produced exophthalmos. They are exceedingly vascular, and the slightest interference with them causes profuse bleeding.

Other growths of the nasal cavities include enchondromata, osteomata, exostoses, adenomata, cystomata, angiomata, and the malignant growths.

Functional Disturbances.

Olfactory Disorders.—The sense of smell may be altered pathologically in the following directions :

a. Diminution (Anosmia).
b. Increase (Hyperosmia).
c. Alteration (Parosmia).

The examination of the olfactory sense is rarely part of a clinical investigation, owing mainly to the fact that no reliable methods of examination have been proposed, and the absence of a standard which may be called health. For the present we are obliged to rest content in presenting to the patient certain odorous substances, selecting such as do not give off irritating vapors, as ammonia, and relying upon the patient's statements as to whether the sensation resulting is up to his usually healthy standard. When it comes to the examination of cases in which alterations of the olfactory sense are reported, we are obliged to rely entirely upon the patient's statements, considering them in the same category as subjective symptoms generally.

(*a*) **Anosmia** is common in all diseases of the nasal cavities in which the mucous membrane is involved. It is a prominent feature of acute catarrhal rhinitis, and of atrophic rhinitis. In the former, the profuse nasal secretion prevents the odoriferous particles from coming in contact with the mucous membrane, and the localized inflammation destroys the sensitiveness of the olfactory terminal fibres. In chronic hypertrophic rhinitis changes in the terminal nerve-filaments are probably the most important factors, as they are, also, in atrophic rhinitis. In the latter condition, the nerve-fibres undergo such extensive degeneration as to make it impossible for the patient to perceive the offensive odor emanating from him.

Anosmia may also find its origin in purely nervous lesions. These include local brain lesions, as tumor, hæmorrhage, localized softening, primary degeneration of the olfactory bulbs and tracts, olfactory neuritis, olfactory nerve atrophy, hysterical disturbances, and congenital defects. The frequency with which olfactory disturbances accompany these several affections is not known, because associated symptoms are generally so obtrusive as to lead to ignoring of the olfactory sense in the clinical investigation of the case. Anosmia from nervous causes may come on suddenly or slowly; it may be preceded by irritative phenomena, *i.e.*, by hyperosmia or olfactory hyperæsthesia, or by parosmia.

Anosmia from disease of the brain may be due to direct implication of the bulb in the morbid process, or it may be produced by pressure acting from a distance, as in cases of tumor of the centrum ovale. Again, the disturbance in cases of brain tumor may originate in an olfactory neuritis, comparable in every way to optic neuritis, which is so generally regarded as an important diagnostic factor in the recognition of intracranial growths. It is believed, also, that olfactory disturbance may originate in cortical disease, the lesion involving the olfactory cortical centre, evidence at hand showing this to be the tip and median aspect of the temporal lobes.

Olfactory neuritis, there is every reason to believe, may originate in any of the causes which may give rise to a similar condition of the optic and auditory nerves. Thus, it may be symptomatic in tumor, meningitis, and certain intoxications,—as syphilis. Most cases of anosmia of neuritic origin are probably preceded by a stage in which hyperosmia is a feature.

Anosmia from nerve atrophy is rarely recorded, though, judging by analogy, a common feature of some diseases. There is every reason to believe that, were examinations more carefully conducted, it would be found in many cases of locomotor ataxia.

The loss of smell in the aged is probably symptomatic of atrophy of the olfactory nerve-fibres.

Hysterical anosmia has been described in association with hemianæsthesia from the same cause. In such cases, the loss of smell is usually on the same side of the body as the remaining sensory disturbances.

Secondary atrophy of the olfactory nerve may result from neuritis, just as may atrophy of the optic nerve follow optic neuritis.

(*b*) **Hyperosmia** or **olfactory hyperæsthesia** may originate in irritative lesions involving any portion of the olfactory sense apparatus. Thus, it may constitute one of the phenomena of lesions above enumerated. It is one of the phenomena of hysteria, in addition.

(*c*) **Parosmia** is a common symptom of irritative lesions, such as tumors or inflammation involving the olfactory sense organs. It is observed clinically with especial frequency in connection with cases of insanity, many cases of melancholia, paresis, etc., being characterized at their onset by olfactory hallucinations. In epilepsy, these sensory disturbances may constitute the aura. A special variety of parosmia has been described in which the patient experiences disgusting odors, and is known as cacosmia. Perversion of the sense of smell is not uncommon in hysterical subjects.

Common sensation of the nasal cavities is occasionally disturbed. There may be anæsthesia, hyperæsthesia or paræsthesia. Anæsthesia of the nasal mucous membrane is a very rarely observed condition, and occurs mainly in association with disease of the fifth pair of nerves. It will always be associated with anæsthesia in other localities. The sense of smell remains undisturbed excepting there be vaso-motor disturbances with excessive secretion, in which case there may be a partial anosmia.

Hyperæsthesia of the nasal mucous membrane is common in a number of acute and chronic inflammatory affections. As a purely neurotic symptom, it is observed almost exclusively in hysteria.

Paræsthesiæ of the nose are comparatively common, though rarely persistent. They are manifestations of hysteria and insanity. Occasionally they constitute the sole symptom. The sensations complained of include a sense of tickling, or stuffiness, or a feeling as of a foreign body or obstruction of some kind.

Sneezing may be said to be a nasal neurosis, when pathological. Under normal conditions, the act is excited when dust or other foreign matter enters the nose. There are conditions which increase the reflex so that paroxysms of sneezing come on under very slight stimulation, or, indeed, without any stimulation. Some persons will get sneezing paroxysms on looking at a bright light; and I know of one case in which they come on as soon as the dinner-bell rings. Cases have been reported in which sneezing was a very annoying symptom during pregnancy. The majority of cases of abnormal sneezing are found on examination to be the result of local conditions readily removable by mechanical treatment.

The Nose in its Relation to Other Organs.—Much has been claimed by rhinologists as to the influence of the nose in the production of disease in other portions of the body. Unprejudiced examination of the subject demonstrates that while the nose is an important disease-producing organ, its importance is nothing like as great as the rhinologist claims. Still it is important enough to call for thorough examination in a very important class of cases. Asthma, in particular, seems to be excited by intra-nasal conditions, and may be cured by removal of the same. Many cases of epilepsy have been reported as cured by rhinologists; but so far as my study of the subject goes, the claims made are altogether too sweeping. Possibly an occasional case of epilepsy, one in many thousands, is so cured. To one experienced in the observation of this dread disease, the many fads for its cures, as advocated by a diversity of specialists, savor of the veriest nonsense, and remind us of the fairy tales of yore.

There is no doubt that the eye may suffer reflexly from nasal disease; the especial symptoms resulting in this connection being asthenopia, conjunctival irritation, keratitis, and lachrymation.

Nasal disorders most unquestionably bear an important relation to the sexual apparatus. Mackenzie, of Baltimore, has studied this subject carefully, and has formulated the following propositions:

1. In some women with healthy nares, congestion of the nasal cavernous tissue takes place during menstruation.
2. Nasal swelling may take the place of the menses.
3. The nasal mucous membrane becomes more susceptible to reflex-producing impressions during menstruation.
4. These conditions are also found during pregnancy at the periods corresponding to the menses.
5. Epistaxis may occur as a vicarious menstruation.
6. There is sympathy between the erectile tissue of the genital tract and the erectile tissues of other portions of the body.
7. Pathological conditions in any one may react on the other.
8. Coryza may be caused by excessive venery.
9. Nasal discharges may be offensive only during the menses.
10. Utero-ovarian disease may exert an important influence over nasal catarrh.

Nasal Obstruction.—From what has been said in the preceding pages, the agencies which may give rise to nasal obstruction may be surmised. It is well, however, to repeat them at this time, that they may be seen at a glance. They include

(a) Swelling of the nasal mucous membrane.
(b) Enlargement of the turbinated bodies.
(c) Deflection of the septum.
(d) Polypi and other tumors.
(e) Synechiæ.
(f) Foreign bodies.
(g) Parasites.
(h) Post-nasal adenoids.

Nasal obstruction should always be regarded as a very serious matter, especially in the young. The deficient aëration resulting from the imperfect respiration produces impaired nutrition. This is seen especially in cases of post-nasal adenoids. The long-continued mouth-breathing leads to deformities of the mouth, to say nothing of the semi-idiotic facial expression engendered by the half-open mouth. Owing to the increased effort required for inspiration, mouth-breathing children often take on thoracic deformities, of which the so-called pigeon-breast is the chief. Lastly, but by no means the least, is the condition known as aprosexia, which is characterized by inability to fix the attention, apparent mental enfeeblement, and general mental malnutrition. Many times I have seen this condition disappear on the relief of nasal obstruction, but especially after the removal of post-nasal adenoids. These children are often believed to be naturally stupid or semi-idiotic, when in reality the nervous system is involved only as a secondary condition, the nasal obstruction being primary.

It is not unusual, also, to find post-nasal adenoids the explanation for frequent recurrences of "colds" in children of apparently lymphatic constitution. Perhaps the word "recurrence" is misapplied, for it may be truly said that these subjects are scarcely ever without a cold, the condition being practically continuous.

The deficient oxidation arising from the obstructed respiration sometimes leads to urinary deposits of uric acid, and the inevitable though fashionable diagnosis of lithæmia.

Personally, I believe that this lesion is a more important reflex cause of symptoms than all the other alleged reflex causes combined. When neglected in childhood, they may be the foundation for aural and pharyngeal difficulties in later years.

Pain in Nasal Diseases.—Pain is, as a rule, not a prominent feature of nasal diseases. In acute catarrhal rhinitis the main discomfort arises from the sense of stuffiness, as is also the case in chronic catarrhal and atrophic rhinitis. In the acute variety there is very frequently severe frontal headache, owing to involvement of the frontal sinuses. Sinus disease is attended with pain varying in severity according to the tension under which discharges are retained in the affected cavity. Pain extending from the throat into the ear is present when the catarrhal process has invaded the Eustachian tubes. Lupus nodules do not by their presence or growth cause pain, although they are sensitive to the manipulation of the probe. Tubercular rhinitis is painless. The principal affections of the nose painful in character include glanders, foreign bodies in the nasal passages, primary syphilis, malignant disease and œdematous rhinitis. The history of the case and the microscopic examination of the discharges establish the diagnosis in glanders. Foreign bodies are not necessarily painful, though the suffering is at times severe, especially when the larvæ of flies have been deposited. Carcinoma in this locality, though painful, is not usually as painful as are similar lesions in other portions of the body.

Cough.—This is not a prominent nasal symptom. When it is associated with nasal diseases it is produced by the irritation of secretions dropping from the post-nasal space into the pharynx. It may arise, however, by reflex irritation of the turbinated bodies and the septal mucous membrane. A dry, tickling cough is sometimes observed in intumescent rhinitis; but, inasmuch as this affection is often associated with some alteration of voice, it is more than likely that the cough is due to an associated laryngeal irritation. A reflex nasal cough has been described by Mackenzie and others. It is due to irritation of certain portions of the nasal mucous membrane, known as cough areas. It is found in association with septal thickenings, turbinated hypertrophies, and local or diffuse congestions and inflammations. It is by no means invariably present, however, in cases of these affections, and yet it is sufficiently frequent to demand careful examination of the nose in cases of cough not explainable on other hypotheses, and resisting ordinary methods of treattment.

Constitutional symptoms may be associated with nasal affections, but their nature is not such as to give them a diagnostic value. Thus in acute catarrhal rhinitis there may be associated some slight rise of temperature, and sensations of creepiness or chilliness, with lassitude and general aching. The obstructed breathing of various chronic affections is associated with the malnutrition that necessarily follows imperfect respiration.

Glanders, diphtheria and measles affect the nose, and are associated with marked constitutional disturbances, which, however, are identical with the same class of symptoms as occur in all cases of these disorders.

CHAPTER X.

THE LARYNX.

Examination of the Larynx.

Apparatus Required.—The apparatus necessary for examination of the larynx include a means for illumination, a reflecting head-mirror, several laryngeal mirrors, and a laryngeal probe.

The Light.—Any kind of light may be employed for the illumination of the larynx when the observer is an adept in the art of laryngoscopy. Still, skillful as well as less-practised hands find special means for illumination necessary to accuracy. Other things being equal, a white light is probably the best, as tending less to alter the actual color of the parts inspected. Hence the electric, the Welsbach incandescent and bright sunlight have their respective advantages. In the case of the first two named, ground-glass globes, by diffusing the light, add to their practicability. As to the sunlight, all of us should accustom ourselves to its use as well as to the diffuse daylight, for circumstances must arise in which artificial light is unavailable.

For office use, numerous devices in the shape of gas- and lamp-brackets having universal movements have been suggested. Any of these is good, providing the bracket is freely movable in all directions, and providing the light carried (Argand-gas-light, incandescent electric with ground-glass, Welsbach, or oil-lamp with small circular wick) is a good one. Chimneys with reflectors and bulls'-eye condensing lenses have been suggested as a valuable accessory for concentrating the light, and are no doubt useful; most observers, however, find them unnecessary. They may therefore be dispensed with as only adding to the numerous paraphernalia about the office.

The Head-Mirror.—This is a circular concave mirror having a diameter of three-and-a-half inches, and a focus of fourteen inches. At its centre is a small, round perforation. Some recommend that the perforation be oval. This suggestion is not a practical one; for the mirror being freely movable on the head, and its position being altered from time to time in the course of the examination, destroys any theoretical advantage to be derived from an oval perforation. The mirror is held on the head by one of the numerous devices. The best is probably a broad belt of webbing with padded metal-shield front to which the mirror is directly attached; other suggestions include spectacle-frames and specially-constructed springs. The joint uniting the mirror with the retaining-apparatus should be of the ball-and-socket variety and unhampered, so as to be freely movable in all directions. At the same time, it must be so constructed that it can be retained permanently in any position desired.

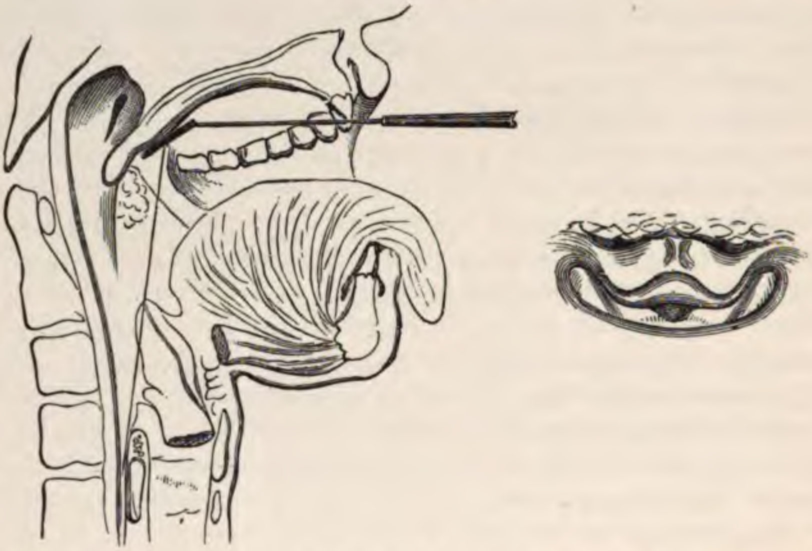
The Laryngeal Mirrors.—These consist of small circular mirrors, ranging in size from one-half inch to an inch-and-a-quarter in diameter, and affixed to a shank-handle at an angle of 130° to 135°. Some prefer mirrors attached to a shank only. When used, they are to be fastened to a universal handle. Such a device has the advantages of portability and some slight economy, but not of convenience in office work.

The Probe.—The probe has but limited utility in laryngeal diagnosis, as it can only aid in determining the consistence of tumors and swellings, the true nature of which is not rendered clear on inspection. As most of such cases should be treated only by physicians possessed of more than ordinary technical skill, it may be said that the laryngeal probe finds no place in the hands of the general practitioner.

Method in Laryngeal Examination.—Accuracy of result is to be secured only by attention to minute details as to position of patient and operator, the light, and the manipulation of instruments. Even then, the examination will be barren of results unless the physician has a proper conception of the normal parts, and then systematically looks over each of these to discover abnormalities of structure and function. It is a very common mistake on the part of the beginner to take no note of lesions of the mouth, soft palate, tonsils and pharynx, before proceeding with his laryngoscopic examination. It must be remembered that laryngeal lesions of certain diseases present most confusing resemblances to each other,—so great, in fact, as to require in very many instances all the aid we can derive from the objective appearances of other parts than the larynx, as well as from the history of the case and the general condition of the patient. A knowledge of information of this character is advisable before examining the larynx, because it aids to a quicker interpretation of what is observed. At the same time, any preconceived notions thus formed concerning the case should not lead to carelessness in laryngoscopy. Special chairs are useless. The patient is to be seated opposite the operator, with the illumination to one side and on about a level with the ear. As to the side at which the light is to be placed, there are different tastes. Most prefer the light to be to the left of the patient, while some claim that the opposite side renders less likely any interference with the illumination by the examining right hand of the physician. The patient is directed to throw his head back somewhat and protrude his tongue; then, with the head-mirror, the fauces are well-illuminated. Now the tongue is grasped between the forefinger and thumb of the left hand by the operator, and gentle traction made upon that organ. Due care must be taken not to injure the tongue by too much pressure on the frænum by the edges of the lower incisor teeth. A laryngeal mirror of the largest size, adapted to the throat under examination, should be selected and well warmed over the flame, the face of the mirror being next to the source of heat. It is then introduced in the pharynx, with its reflecting surface downwards. The handle of the mirror should be grasped as a pen-holder. If the above manipulations are properly performed, the laryngeal image is brought into view.

In order to secure a firm grasp of the tongue, a small napkin or doiley should first be folded about the tip of the organ. If a patient objects to these, thinking of the possible mouths in which they have been used previously, a strip of gauze bandage may be employed. In order to let both hands of the operator be free, the patient may be taught to hold his tongue himself,—although, for examination purposes alone, this is rarely necessary.

FIGS. 50 and 51.



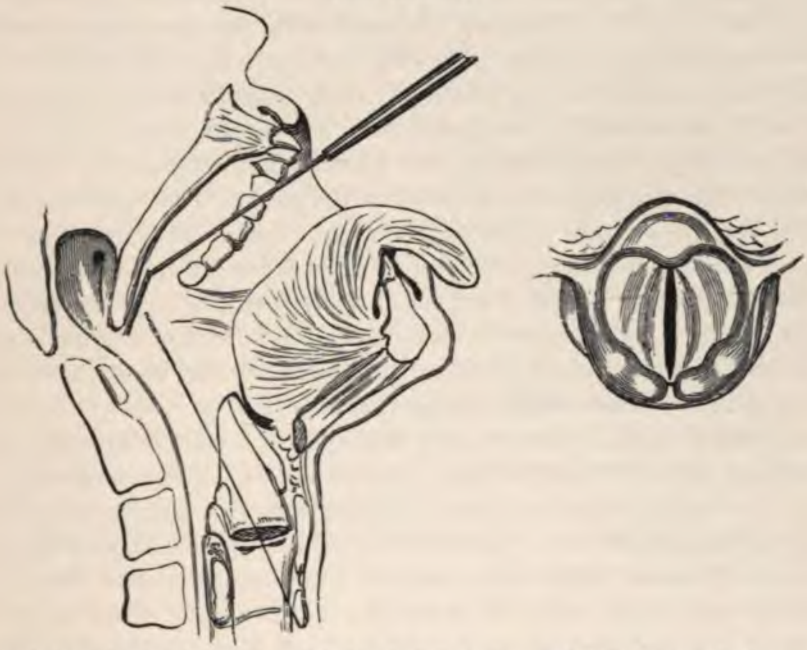
Sectional view, showing the position of the head of the patient and of the laryngeal mirror, which will give the minimum amount of view. The laryngoscopic image in such a case is represented in the smaller figure at the side, 51.

Proper warming of the laryngeal mirror is important, because, if the cold instrument is inserted, the moisture of the breath will at once condense on its surface and fog the same, making it impossible to obtain a clear image. After warming the mirror, its temperature should always be tested on the operator's hand, lest it be too hot for the patient's welfare or too cool for the purpose desired.

As large a mirror as can be skillfully introduced is advisable, because it will afford a view over a larger surface. Such mirrors, however, may be resented when the fauces are contracted or are unduly irritable.

On introducing the mirror, it should be carried through the mouth without touching any of the parts until it enters the fauces, when its posterior surface should rest against the soft palate and uvula, pushing the latter backwards and somewhat upwards. Care must be taken not to permit its edge to touch the posterior wall of the pharynx. If the laryngeal image does not come at once into view, the hand holding the mirror should be raised or the handle of the mirror rotated slightly until the proper position is obtained. Throughout these manipulations, the hand must be kept to one side and out of the line of vision.

FIGS. 52 and 53.



Sectional view, showing the position of the head of the patient and of the laryngeal mirror, which will give a full amount of view. The laryngoscopic image in such a case is represented in the smaller figure at the side, 53. This and Figs. 50 and 51 after Lennox Browne.

Errors in Laryngoscopic Technique.—Although laryngoscopy is a very simple process, failures to obtain satisfactory views of the larynx are not uncommon with beginners. These arise from one of the following causes: 1, undue haste; 2, imperfect illumination; 3, clumsiness in manipulation of instruments or in holding the tongue; 4, officiousness of manner, thus getting the patient nervous and excited; 5, excessive irritability of the fauces; 6, the dorsum of the tongue is not sufficiently depressed; 7, enlarged tonsils; 8, pendulous epiglottis.

It is necessary to bear in mind that, excepting in the hands of those of wide experience, the laryngoscope does not give information to be taken in at a glance. Each part to be shortly mentioned must be carefully inspected, and its functional condition determined. It must also be remembered that, if the image does not spring into view at once, the angle of the mirror to the vertical must be altered until a view is obtained. It is important that the fauces be properly illuminated before the mirror is introduced, and the operator trained in maintaining the light in that position. It is always better to have the head-mirror so placed that the operator shall look through its central perforation. If the first attempt does not meet with success, it is wise to withdraw the mirror and make a second one in a few seconds or so. The irritability of the fauces in some patients is sufficiently great to make satisfactory examination impossible, although the skill of the operator in handling his instruments may have some influence in lessening this evil. It may be readily overcome, however, by having the patient suck lumps of ice for fifteen or twenty minutes before the examination, or by anæsthetizing the fauces with a two per cent. cocaine spray. Habit is a very important factor here, as most persons lose their faucial irritability after repeated manipulations of the parts. The up-riding of the tongue is to be obviated by having the patient take a deep breath, or hold his own tongue while the operator pushes the dorsum down with a tongue-depressor. Failure arising from tonsillar hypertrophy is to be overcome only by using a small mirror. Sometimes this source of trouble is insurmountable. If the uvula is elongated and obstructs the view, then a large mirror should be used, and so placed that it pushes the uvula to one side or supports it above. The difficulty arising from a pendulous epiglottis is not always overcome with facility. Sometimes the mere pronunciation of "*ee*," or coughing with the mirror *in situ*, is sufficient to raise the part and bring the larynx into view. In still other cases it is necessary to direct the patient to throw his head well backwards and bring the mirror close to the posterior pharyngeal wall, and somewhat more vertical than usual. At the same time the observer keeps his eyes well above the level of the patient's mouth. In some cases all of these devices fail, and then it is necessary to use a retractor to raise the epiglottis.

The method of laryngoscopy as above described affords a view of the anterior wall of the larynx. Killian has offered an invaluable suggestion for inspection of the posterior wall of the organ and the posterior ends of the cords and underneath their surface. The patient is made to stand, with his head bent well forward. The examiner kneels and holds the mirror well up against the uvula. By this device the posterior wall of the larynx and the trachea as far as the bifurcation may be inspected. This method is not very easy of execution, as the position of the examiner is far from comfortable, and the light does not come from a suitable direction.

Autoscopy of the larynx is a modern adaptation of the oldest method of laryngoscopy. The patient is seated, with chest thrown forward and head bent somewhat backwards. A specially-constructed tongue-mirror with electric-light attachment is employed to draw the tongue very much forward, when the examiner can look directly into the larynx and see its posterior part, the arytenoids, and sometimes the posterior portion of the cords and part of the trachea. The method is advised as especially applicable to children under anæsthesia. Of late, Kirstein, to whom the modern application of the instrument method is due, has dispensed with the autoscope, and in its place uses a specially-constructed tongue-spatula. "For the sake of description, we distinguish between the tongue portion of the instrument and the handle. The distal end of the tongue part, for a distance of 5 cm., is bent downward, the amount of this curve being equal to 1 cm. In other words, if looked at in a profile view, the distal 5 cm. form the segment of a circle, with a radius of 13.5 cm., to which the proximal straight portion of the spatula forms a tangent. At its free distal extremity there is a shallow notch, so as to admit of its more ready adaptation or approximation against the median glosso-epiglottic ligament." Kirstein lays considerable stress upon the exact amount of curvature of this part of the instrument. If the curve is insufficient, autoscopy of the larynx in many cases is not successful when it would succeed with a more suitable instrument, because the pressure on the tongue necessary for the raising of the epiglottis fails. On the other hand, if the bend be too pronounced, the spatula is useless. He gives the following measurements, which appear to satisfy the requirements: "The length of the tongue portion from the notched extremity to the handle-shoulder is about 11 cm., the breadth is about 15 mm., and the thickness about 3 mm. The edges of the spatula must be well rounded."

The Laryngeal Image.—With proper examination-technique the following structures are rendered visible: The epiglottis, superior and lateral glosso-epiglottic folds, the valleculæ, the vocal cords, the chink of the glottis, the anterior wall of the trachea, the bifurcation of the trachea (in *exceptionally* favorable cases), the ventricular bands, the ventricle of Morgagni, the arytenoid cartilages, the interarytenoid space, the aryteno-epiglottidean folds, the cartilages of Wrisberg and Santorini, and the pyriform sinuses.

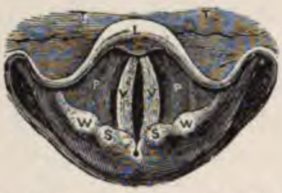
An extensive anatomical description of these is out of place here, but their location and relations may be readily understood by consulting the annexed diagrams (p. 340), which give a good conception of the positions of the vocal cords in the production of tones from the lowest to the highest.

What to Observe.—When making an examination, special attention should be paid to the following points: 1, the color of the laryngeal mucous membrance; 2, the form and contour of the various parts of the larynx; and, 3, the functional activity of the vocal cords during phonation and respiration.

Each of these factors is to be studied, bearing in mind the anatomical structures entering into the composition of the larynx and its functions. Clinically, the larynx, it must be remembered, consists of mucous membrane, cartilages, connective tissue, articulations, muscles, nerves, and vocal cords, so constructed as to constitute the organ for voice-production, and a most important portion of the respiratory tract. If, then, its structure is sufficiently altered by disease to disturb its functions, the results are disordered voice or difficult respiration, or both.

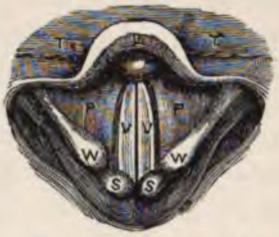
Knowing, then, the anatomical structures making up the larynx, we are able to state that its diseases are those peculiar to the individual tissues above mentioned, and these are to be summarized as follows:

FIG. 54.



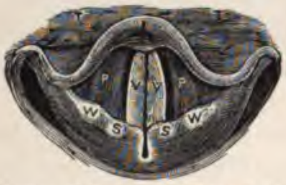
Laryngeal image. T T, tongue; P P, ventricular bands; L, epiglottis; S S, cartilages of Santorini; V V, vocal cords; W W, cartilages of Wrisberg.—After Lennox Browne.

FIG. 55.



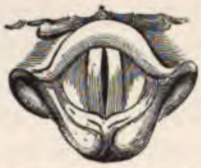
Laryngeal image. T T, tongue; P P. ventricular bands; L, epiglottis; S S, cartilages of Santorini; V V, vocal cords; W W, cartilages of Wrisberg.—After Lennox Browne.

FIG. 56.



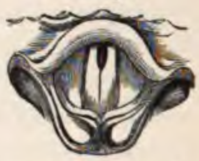
Laryngeal image. T T, tongue; P P, ventricular bands; L, epiglottis; S S, cartilages of Santorini; W W, cartilages of Wrisberg.—After Lennox Browne.

FIG. 57.



Laryngeal image.—After Lennox Browne.

FIG. 58.



Laryngeal image.—After Lennox Browne.

1. **Mucous membranes,** including subjacent connective tissue.
   a. Anæmia.
   b. Active hyperæmia.
   c. Passive hyperæmia.
   d. Hæmorrhages and hæmorrhagic extravasations.
   e. Œdema.
   f. Acute catarrhal inflammation.
   g. Chronic catarrhal inflammation.

2. **Cartilages.**
    a. Perichondritis.
    b. Necrosis or abscess resulting from (a).
3. **Articulations.**
    a. Rheumatism.
    b. Anchylosis.
4. **Nerves.**
    a. Paralysis.
    b. Spasm.
        Involving the distribution of the
            Superior laryngeal nerves, or
            Recurrent laryngeal nerves.
5. **Muscles.**
    a. Rheumatism.
    b. Under- or over-action, according to the presence of one or the other of the nervous disturbances above mentioned. These disturbances of the nerves and muscles are exhibited mainly in alterations in the movements of
6. **The vocal cords,** which, being covered by mucous membrane, are also liable to the various pathological conditions of that tissue.
7. **Any of the laryngeal tissues** is liable to
    a. Ulcerations, which may be
        i. Simple.
        ii. Syphilitic.
        iii. Tubercular.
        iv. Malignant.
        v. Lupoid.
    b. Tumors.
        i. Papilloma.
        ii. Fibroma.
        iii. Carcinoma.
        iv. Mucous polypi from cystic dilatation of glands.

Symptoms of these various lesions as occurring in the larynx must include—

1. Symptoms of the pathological conditions *per se.*
2. Symptoms of these pathological conditions as modified by the peculiar construction of the larynx.
3. Symptoms pointing to deranged laryngeal function.

After this digression, let us return to a study of the pathological conditions evident on laryngoscopy. I said that the color of the mucous membrane should be noted. Normally, the laryngeal mucous membrane is a pale pink or red; the epiglottis exhibits a slightly yellowish hue; and the vocal cords a pearly white, bordering somewhat on the pinkish. The healthy parts are, however, very susceptible to slight influences; hence it is not unusual to find the normal redness increase in density after the slight manipulations of examination. A knowledge of this fact is necessary, to avoid errors.

1. **Changes in the Color of the Mucous Membrane.**—An increased redness is observed in any condition producing hyperæmia or inflammation of the mucous membrane. The differentiation of these two processes is made by the recognition of slight puffiness or swelling in the latter. In the majority of cases it is a simple catarrhal laryngitis; in acute cases it is almost certainly so. This fact, however, should not deter the observer from searching for other lesions, especially in chronic cases,—bearing in mind that they may be found when least expected. The mucous membrane inflammation may be an associated condition of one of the acute infections,—notably of typhoid fever, measles, and variola. It may be the initial stage of a chronic infection, namely, syphilis. It may have originated in the habits of the individual, as excessive indulgence in tobacco. It may be due to the inhalation of irritating vapors or of a dust-laden atmosphere.

A pale, lustreless, grayish color of the laryngeal mucous membrane is sometimes seen in old cases of catarrhal inflammation. Such a state is due to atrophy of the structures.

Chronically inflamed laryngeal mucous membrane with a constant tendency to relapse, and accompanied by ragged ulceration of the vocal cords, is strongly presumptive of syphilis. It is especially liable to occur during the interval between the secondary and tertiary stages of that disease.

A slight, persistent, unilateral congestion appearing on one vocal cord in a person past the age of forty may mark the beginning of a malignant growth.

Passive hyperæmia of the larynx may be observed in conjunction with uncompensated heart lesions, and in cases of intra-thoracic tumors.

The redness may invade the mucous membrane as a whole, or be limited to parts of the same. Circumscribed redness seems to be more common in the chronic catarrhs.

Grayish-red or whitish-yellow elevations surrounded by an inflammatory areola suggest mucous patches of syphilis. These lesions are exceedingly rare; indeed, some laryngologists deny their existence.

Localized anæmia of the larynx without evidences of anæmia of the external surface must be regarded as suggestive of the possible future advent of laryngeal tuberculosis, especially if associated with a similar condition of the pharynx. Pallor of the laryngeal mucous membrane is also observed in chlorosis, pernicious anæmia, leukæmia, and secondary anæmias. Here associated conditions establish the true character of the case.

Discharges on the Laryngeal Mucous Membrane.—A scanty mucous or muco-purulent discharge may be observed on the mucous membrane in cases of acute or chronic catarrhal laryngitis, whether primary or secondary.

Hæmorrhagic erosions may occur in conjunction with inflammatory processes of high grade. Submucous extravasations have been observed as a result of asphyxia, purpura, and other constitutional hæmorrhage-producing affections, *e.g.*, the essential anæmias. Actual hæmorrhage points indisputably to an opening into a blood-vessel, as from ulceration, tumor, or vascular degeneration.

2. **Structural Alterations.**—These include tumefaction and other changes in the soft parts without abrasion of the mucous surface, abrasions and ulcerations, abscess, œdema, tumors, malformations, and foreign bodies.

(*a*) **Tumefactions.**—Generalized and localized swelling of the mucous membrane is observed in connection with catarrhal inflammation. Indeed, as already stated, it constitutes the change which enables one to differentiate a simple hyperæmia from actual inflammation. It may be limited to individual parts or structures of the larynx. Its recognition, unless exceedingly well-marked, demands that the observer be thoroughly acquainted with the contour of the normal larynx. Especially is this true when the swelling is evenly distributed. When localized, it may be almost nodular. Sometimes it is rough and uneven, owing to enlargement of the papillæ and glands. Such conditions are especially liable to occur in connection with long-standing catarrhs. They are almost always associated with similar changes in the pharynx. Sometimes the thickening of the mucous membranes is so great as to constitute polypoid or nodular excrescences, which conditions will be mentioned shortly, when treating of tumors.

When the thickening involves the vocal cords, these bodies appear as if they had a well-marked rounded swelling on their upper surfaces. In other cases the edges of the cords are uneven, and may give the appearance to careless observers of actual loss of tissue. These changes are found in chronic laryngeal catarrh.

Sometimes both true and false vocal cords present a granular appearance, due to the formation of nodules of varying size. This condition is believed to be the result of a hyperplasia of the epithelium, in conjunction with hypertrophy of the submucous tissues. It occurs only in the impoverished and badly-nourished, living under bad hygienic conditions. It is regarded as the local counterpart of granular ophthalmia, and hence has been designated trachoma of the cords.

(*b*) **Abrasions and Ulcerations.**—When studying ulcerations of the larynx, note their number, size and location, the character of their surfaces, edges and areola, and study the facts thus observed in connection with the rest of the laryngeal and constitutional symptoms of the case.

Simple abrasion of surface undoubtedly occurs in acute and chronic laryngeal catarrhs. The mucous membrane, delicate as it is in the normal state, is even more so when inflamed. It is to be expected, then, that the acts of coughing and clearing the larynx of secretions under such circumstances must work some injury. In the majority of instances, the abrasions are too slight to be detected by the laryngoscope.

The **true ulcerations** include those of syphilis, tuberculosis, and lupus. Ulceration from syphilis may occur during either the secondary or tertiary stages of the disease. Those of the secondary stage are generally superficial, and not infrequently associated with the cutaneous lesions of that period; very often they are multiple. Sometimes they extend and coalesce, forming a large ulcer, which presents a grayish base and well-defined edges, and, on healing, a depressed cicatrix, which is regarded as characteristic.

Superficial erosions of the vocal cords, posterior surface of the epiglottis, and ventricular bands, associated with a brownish mottled appearance of the vocal cords, occur in the secondary stage of syphilis.

Tertiary ulceration is generally indicated by a single lesion which may occur in any part of the larynx, though more frequently situated in the upper portion of the organ. It frequently involves the epiglottis, the entire substance of which may be destroyed. Another not infrequent location for the ulceration is along the aryepiglottic fold to the ventricular band. Very characteristic of all tertiary syphilitic ulcerations of the larynx is their tendency to the production of deformities, owing to their peculiar cicatrices.

Tubercular Ulcerations.—Tubercular ulcers are generally multiple, and are of much slower development than the syphilitic variety. They are generally preceded by a pale, lustreless, opaque thickening of the mucous membrane. Their favorite location is in the lower and posterior portions of the larynx. As they do not heal, there is no tendency to cicatrix formation; and for this reason, and the additional one of their superficial depth, deformities are unlikely, no matter how widespread the destruction of tissue. Their true nature is established by the associated tubercular disease of the lungs and the discovery of Koch's bacillus in the discharges.

Lupoid ulcerations are rarely if ever observed in the larynx, excepting in connection with similar ulcerations elsewhere, *e.g.*, on the skin and in the pharynx. Of course this fact avails us little if, as sometimes happens, the lupoid ulceration is located in the larynx primarily. It resembles the tubercular ulceration more closely than any other variety,—indeed, may be indistinguishable from it. Lupus shows a very slowly progressive tendency, advancing in some directions, healing in others. Hence, some cicatrization is observed. Nodular formations characterize the earlier stages of the pathological process, but, owing to the absence of local symptoms, may escape discovery. These nodules soften, become what has been described as of "an apple-jelly appearance," and then ulcerate. Like tubercular ulcers, we find a worm-eaten appearance. Unlike tubercular processes, distortion is often a prominent feature. Cicatricial stenosis and dyspnœa often arise. When it attacks the epiglottis, it is the free margin of that structure which is invaded. The vocal cords are usually the last part to be affected.

Tabular Differentiation of Syphilitic and Tubercular Ulceration of the Larynx.

	SYPHILITIC.	TUBERCULAR.
Associated conditions.	Syphilitic lesions elsewhere; history of infection.	Pulmonary tuberculosis.
Number of ulcers.	Usually single.	Generally multiple.
Preceding mucous membrane changes.	Localized unilateral swellings, or by gummatous growths.	Peculiar lustreless, opaque thickening.
Size.	Generally larger than tubercular ulcers.	
Favorite locations.	Anterior surface and free edge of epiglottis; upper portion of the larynx.	Lower and posterior portions of the larynx and ventricular bands; lower and anterior surface of epiglottis.
Symmetry.	Generally unilateral.	Bilateral ulceration favors tuberculosis.
Deformity.	Great and varied.	Absent or slight.
Appearance of ulcer.	Deep, cleanly cut, well-defined shelving walls; inflammatory areolæ, extending in depth.	Surrounded by anæmic mucous membrane; more shallow; worm-eaten appearance; spreads laterally or in serpiginous manner.
Secretions.	Relatively scant; slow accumulation. Fœtid odor.	Profuse; accumulates with rapidity; contains tubercle bacilli. Peculiar sweetish odor.
Hæmorrhage.	Rare.	Common.
Pain.	Slight or absent.	Severe.
Respiration and voice.	Unless vocal cords are involved, voice not impaired.	Respiration embarrassed and voice feeble from associated chest condition.
Cicatrices.	Stellar; cicatrices observed on pharynx and elsewhere. Start from edge of ulcer.	No tendency to cicatrization.

Laryngeal leprosy, with ulceration, is a rarely-observed condition. It almost never occurs until sufficient time has elapsed to render the cutaneous lesions sufficiently well-marked to make the diagnosis unquestionable, aside from the laryngeal condition. As in lupus, the ulceration is preceded by formation of nodules, which form the basis for the disintegration of tissue. The ulcers are at first small and rounded, and elevated above the contiguous mucous membrane. They have been compared by De la Sota to syphilitic mucous patches. They are apt to extend in depth and involve the cartilages. Here it bears a resemblance to the tertiary syphilitic ulcerations. From mucous patches the lesions are to be differentiated by their grayish-red color. Leprous patches are apt to be anæsthetic; syphilitic, the reverse. Finally, we have the therapeutic test, which should be tried in every doubtful case before coming to a positive conclusion.

The **ulceration of malignant disease** presents no differential diagnostic features. Indeed, such are rarely necessary, for the tumor usually makes its presence known before the ulcerative stage has been reached. In the case of carcinoma, the disintegration of tissue is due to the abundance of cell proliferation, rather than to friction of the parts. The excavations are fissured or crater-like, especially in encephaloid cancer. In epithelioma the ulcerations are more superficial. Glandular involvement is rare; still, when it does occur, it does so early in the progress of the case. Sarcoma frequently exhibits points of erosion; ulceration seldom occurs.

(*c*) **Abscess.**—Abscess here, as in other localities, shows itself as a rounded, symmetrical, fluctuating tumor. It must always have been produced by infection by the bacteria of suppuration, and hence is secondary to other conditions, as perichondritis, foreign bodies, or malignant disease. The preceding history, and especially the progress of the case, constitute important diagnostic factors.

(*d*) **Œdema.**—As ordinarily employed, the term "œdema of the larynx" is restricted to a condition in which œdema is practically the only local symptom. It is exhibited, as a rule, in three distinct serous sacs, viz.: one starting on the posterior surface of the epiglottis and extending until it involves the glosso-epiglottic fossæ, and the others formed by the distention of the aryepiglottic folds. In the mirror it is seen as three distinct swellings, the mucous membrane covering of which is tense, semi-glassy, and partly transparent. In their midst is a triangular opening, the remains of the glottic chink.

Œdema without other marked local changes occurs most frequently as a manifestation of renal disease. A relaxed state of the mucous membrane, of course, favors its occurrence. It may also be the result of venous obstruction arising from the pressure of an aneurysm or other tumor in the cervical or upper thoracic regions. Angio-neurotic œdema may exceptionally attack the larynx. It is due to a vaso-motor paresis, and occurs in neurotic subjects. It is usually associated with similar changes on the cutaneous surface.

Œdema in conjunction with other local manifestations is to be recognized by the presence of local or diffuse swellings having the appearance above described. They are due, of course, to vascular stasis.

(*e*) **Tumors.**—These may be either malignant or benign. The latter are the more common, and, while existing in great variety, are in practically every case either papilloma, fibroma or cystoma.

Papilloma.—These may be either single or multiple, usually pedunculated, and situated by preference on the vocal cords, especially on their anterior thirds. They never infiltrate.

Fibromata are nearly always single, and may be either sessile or pedunculated. They generally originate in an inflammatory thickening of the fibrous basis of the vocal cords, and are manifested as white, pink, cherry-red or bluish tumors, with a smooth surface. They are generally situated on the anterior half or at about the middle of a vocal cord. The pedicle may be sufficiently long to permit the tumor to drop into the subglottic space, in which case a correct diagnosis may not be made.

Cystomata result from obstruction of the orifices of the muciparous glands, especially on the dorsal surface of the epiglottis. They are shown as smooth, tense, globular, semi-translucent tumors covered with light-red or grayish-pink mucous membrane. Usually small, they may be sufficiently large to seriously embarrass respiration.

Malignant growths include carcinomata and sarcomata. Bosworth uses the term " grumous " as best calculated to describe the appearance of sarcomata, which he says appear as " soft, irregularly-rounded, pinkish, or more often grayish, semi-opaque tumors," distorting the soft parts.

Carcinomata being due to cell infiltration, the extent of the pathological process is always greater than inspection would indicate. This is made manifest by unusual disturbance of function from involvement of the muscles and articulations. The tumor itself is shown as a broadly-infiltrating mass, irregular in outline, distorting the parts, and sooner or later accompanied by ulceration of the character already described. (*Vide supra.*)

Malignant disease in its early stages may appear as a diffuse infiltrating growth with uneven surface, as a dirty-white or whitish-gray oblong wart on one vocal cord, or as an uneven fringe-like outgrowth from the cord.

(*f*) **Malformations of the larynx**, and other minor defects, are not infrequent. They are sufficiently obvious to any one at all acquainted with the normal appearances of the larynx ; and, owing to this and their great variety, a description of them here is not advantageous.

(*g*) **Foreign bodies** are not to be diagnosticated by the laryngoscope unless favorably located. Their discovery, therefore, must in the majority of cases depend upon the history of the case and the associated symptoms. Sometimes, when the body itself is not visible, its location will be suggested by a localized tumefaction, the cause of which is evident from the history of the case.

3. **Functional Activity of the Vocal Cords During Phonation and Respiration.**—In the beginning, let me impress upon the reader, when examining the functional activity of the vocal cords, that he do not neglect their movements during respiration. Attention to this point will save many a diagnostic error.

The functions of the vocal cords may be disturbed by reason of alterations in their structure, and by impairment of the functions of the nerves which innervate their muscles. The larynx derives its nervous supply from two sources,—the superior laryngeal and the recurrent laryngeal nerves. Both are branches of the pneumogastric. The former is a sensory nerve of the larynx, and, in addition, supplies the crico-thyroid and arytenoid muscles. The recurrent laryngeal supplies the remaining muscles of the larynx. This nerve has close anatomical relations with the subclavian artery (on the right side), the aorta, right pulmonary apex, trachea, and œsophagus, which must be borne in mind when attempting to solve many of the clinical problems in which disturbance of its functions enter.

Normal Position and Movements of the Vocal Cords.—When the patient is breathing quietly, the vocal cords should lie midway between the positions of adduction and abduction, converging anteriorly and separated posteriorly. They are wider separated than in the so-called "cadaveric position," because of a persistent reflex. On phonation, the vocal cords come into close or exact apposition in the median line. On deep inspiration, they separate widely at their posterior extremities.

Loss of Motor Functions of the Vocal Cords.—(*a*) **During phonation the vocal cords remain in the position of inspiration.** This shows a paralysis of the crico-arytenoidei lateralis, arytenoidei transversus, and the thyro-arytenoidei internus and externus, or adductor muscles of the cords. This condition is almost always bilateral. It is often functional, as in hysteria, and may occur in conjunction with the general weakness of such diseases as phthisis and anæmia, and it has been observed as one of the phenomena of catarrhal laryngitis from infiltration of the muscles. It is rarely complete. When unilateral it is due to local causes, and may then be due to cold, small-pox, or syphilis. In bilateral paralysis of hysterical origin the onset is sudden; after lasting an indefinite time, the paralysis departs as suddenly as it came. When the trouble arises from other causes, the onset is in keeping with the pathological process producing it.

(*b*) **The anterior extremities of the cords come together during phonation**; the posterior do not;—leaving a small triangular opening in that locality. This shows that the arytenoidei transversi are affected.

(*c*) **The vocal cords come together at both extremities,** *but remain separated at the middle*, forming an oval-shaped chink. The thyro-arytenoidei interni are paralyzed. This condition is often due to overstraining the voice, or to catarrhal laryngitis in anæmic or neurotic subjects.

A similar condition may be produced by improper use of the voice, and is one of the most frequent causes which affect the clearness and carrying qualities of the voice of public speakers, producing what is known as a weak "breathy" or non-resonant tone. The failure of the vocal bands to close allows too much air to escape before the tone is produced.

(*d*) **A Double Elliptic Glottic Chink is Formed During Phonation.**—This signifies paralysis of the arytenoidei transversi and the thyro-arytenoidei interni.

(*e*) **One or both vocal cords are observed in the median line during quiet respiration,** and do not depart therefrom during deep inspiration. There is loss of power of the abductor muscles (crico-arytenoidei postici). This condition may be due to pressure on the recurrent laryngeal nerve of aneurysm or tumor in the neck or thoracic cavity, enlarged mediastinal glands, pleuritic thickening (right side), central nerve lesions, disease of the pneumogastric or spinal accessory at the base of the brain, cerebral syphilis, bulbar paralysis, tumors of the brain, bulbar hæmorrhages or thickening of the dura mater, neuritis from typhoid fever, diphtheria, pneumonia, rheumatism, grippe, lead or arsenic.

(*f*) **During Quiet Respiration, Phonation, and Deep Inspiration, One or Both Vocal Cords Remain Fixed in the Cadaveric Position.**—This shows complete paralysis of one or both recurrent laryngeal nerves. The majority of cases of recurrent laryngeal paralysis are unilateral. When left-sided, aneurysm of the arch of the aorta is suggested. The most frequent cause, however, is disease of the nerve in its course through the neck, being much more exposed on the right than on the left. It may also result from central disease (lesions involving the pyramids, olivary bodies and restiform bodies, and the floor of the fourth ventricle), as in hæmorrhage, endarteritis, disseminated sclerosis, or locomotor ataxia.

Bilateral recurrent palsy is in the vast majority of instances due to cerebral disease. Still, cases have been observed in which it resulted from pericardial effusion.

Consideration of Other than the Laryngoscopic Symptoms of Laryngeal Disease.

1. **Pain.**—Pain is not a prominent feature in the majority of cases in which the larynx is diseased. As a rule, the patient complains of a sensation of mere stuffiness or discomfort. For example, it is not severe in acute catarrhal laryngitis, the patient complaining only of a sense of rawness, burning, or slight cutting. Still, this statement is not of universal application, for severe pain has been experienced in acute laryngitis, especially when occurring in persons of rheumatic diathesis.

It is slight, also, in the majority of cases of laryngeal syphilis. Exceptionally, it may be severe.

It amounts to a mere soreness in most cases of laryngeal lupus.

It is severe and sharp in malignant disease, laryngeal tuberculosis, and perichondritis. In malignant disease, the pain frequently shoots into the ears, being propagated thence by the auricular branches of the pneumogastric.

The majority of laryngeal pains are aggravated by external pressure over the larynx, and during attempts at phonation and deglutition.

2. **Paræsthesiæ.**—These include burning, tickling, itching and other sensations, feeling as of a foreign body in the larynx, and the globus hystericus.

Burning, tickling, itching, pressure, etc., are observed in many cases of catarrhal condition, and in neuroses. A sensation as of a foreign body in the larynx may persist after the foreign body has been removed, either because of the hysterical temperament of the patient or because the foreign body has produced a local tumefaction which still persists. It is also present in many cases of laryngeal œdema.

The globus hystericus consists of a sensation as of a lump or ball rising in the throat, exciting a desire to swallow. It occurs in connection with emotional states of mind in hysterical subjects, and is apt to be associated with numerous hysterical symptoms. It is observed in hysteria, hypochondriasis, and chlorosis.

3. **Hyperæsthesia.**—Laryngeal hyperæsthesia is made evident by the irritability of the larynx to slight excitation. Thus the inhalation of cool air, or phonation, excites a tickling in the organ, with desire to cough. Sometimes the irritability is so great that the mere passage of food over the closed glottis is sufficient to excite spasmodic coughing fully as severe as may be observed when food escapes accidentally into the normal larynx. Laryngeal hyperæsthesia is observed in all diseases of the organ, but is most frequently observed in connection with acute catarrhal laryngitis and in the early stages of phthisis. It sometimes exists as a purely nervous phenomenon, as in hysteria. In infants it may occur from reflex irritation of dentition. Laryngeal hyperæsthesia sometimes attends acute pharyngitis, and also chronic pharyngitis when dependent upon excessive addiction to alcohol.

4. **Anæsthesia.**—Anæsthesia of the larynx may be either functional or organic. When the former, it is due to hysteria. This form is rarely observed in this country, at least. Organic anæsthesia always means a suspension of the functions of the superior laryngeal nerves, and may constitute one of the clinical phenomena of post-diphtheritic paralysis, bulbar paralysis, general paralysis of the insane, tumors of the brain, and locomotor ataxia. It is apt to be attended by paralysis of the muscles innervated by the superior laryngeal nerve. It is recognized by the insensitiveness of the laryngeal mucous membrane to manipulations, as with a probe.

5. **Dysphagia.**—Difficulty in swallowing, when due to laryngeal disease, is the result either of food finding its way into the larynx, causing choking, or of pain produced by the contraction of the laryngeal muscles in closing the glottis; hence it is observed in connection with all destructive ulcerations of the epiglottis, in all cases in which the epiglottis is fixed,

in marked laryngeal hyperæsthesia, and in acute inflammation of the epiglottis. In some cases of laryngeal œdema, difficulty in swallowing is the result of obstruction; in others, food escapes into the larynx from defective closure of the glottis.

The term "swallowing the wrong way" is applied to the escape of food into the larynx during deglutition. In health it cannot occur excepting when a person is off his guard, as he may be when laughing during the act of swallowing. It also occurs when the pharynx and larynx are anæsthetic, owing to the break in the sensory portion of the reflex arc of which the mechanism closing the glottis is the motor-part.

6. **Disturbances in Phonation.**—These may all be included under the one term "aphonia," or loss of voice, of which the various disturbances really constitute different degrees of intensity or severity. Thus, we may note changes in the force of the voice, alterations in its pitch, and changes in its quality. Under the latter head we may also refer to stammering of the vocal cords and diplophonia.

In the beginning, let us emphasize the points of distinction between aphonia, anarthria and aphasia. Aphonia is a simple loss of voice; the patient can pronounce words, but does so with a whispering voice. In anarthria the power of articulation is lost owing to paralysis of the tongue, the organ presiding over this function. Phonation is perfect. In aphasia there is loss of the power of speech from disease of the speech-centres in the brain, the movements of the larynx and tongue being preserved.

Aphonia occurs in all diseases which interfere with the motility of the vocal cords or cartilages to which the same are attached, and in diseases characterized pathologically by swelling of the mucous membrane of the larynx; in other words, conditions which prevent sufficient approximation of the cords, or by changes which diminish the lumen of the larynx and interfere with the column of air passing through that organ. Hence it occurs in connection with the various paralyses and anchyloses of laryngeal cartilages, and in diseases occluding in whole or in part the larynx,—as acute and chronic catarrhs, œdema, tumors, perichondritis, and ulcerations. Sometimes the change in voice amounts to but a simple hoarseness, while in more severe cases the patient cannot speak above a feeble whisper.

In chronic laryngitis the aphonia is usually worse in the morning, the patient almost always regaining a certain amount of power after he has used his voice for a time. Owing to the alterations in the structure of the cords, more muscular effort is required in bringing them into action; hence the voice tires easily and becomes feeble.

In laryngeal tuberculosis the degree of aphonia is sometimes disproportionately severe to the local lesions. Indeed, aphonia may occur in pulmonary tuberculosis independently of any laryngeal involvement. This is explained by the involvement of the right recurrent laryngeal nerve by pleuritic thickening, or by pressure upon one or both recurrent laryngeal nerves by enlarged bronchial glands.

One very interesting variety of aphonia not mentioned in the above is that arising from mental influences. It is well illustrated in the aphonia occurring from anger and other forms of mental excitement; also in hysteria. The latter is characterized by such loss of voice that the patient cannot speak above a whisper, and yet the power to give a normal cough is retained. It comes on suddenly, as a rule, and after lasting an indefinite period, is apt to disappear as rapidly as it came.

Changes in the force of the voice are more likely to depend upon general rather than upon local conditions. Like aphonia, it may be the result of mental influences. Many a patient will consciously or unconsciously speak in feeble tones, and need only be aroused to use the voice with normal force. From substantive disease it is especially apt to occur in all diseases which weaken the general muscular tone, *e.g.*, phthisis without laryngeal involvement, and cholera. It may also be encountered in painful laryngeal diseases, the patient speaking feebly to save discomfort.

The pitch of the voice is due to the rate of the vibrations of the vocal cords. Hence it may be altered by conditions which affect their motor-power, tenseness and thickness, and the force with which air is impelled through the glottis.

Alterations may be produced by structural changes in the laryngeal structures, generally by conditions which influence the tense muscles of the vocal cords, and to inflammatory changes in the mucous membrane covering the cords, and in the cords themselves. The pitch is lowered by conditions which relax the mucous membrane, and weaken the nervous or muscular systems, or makes the tense condition of the vocal cords and consonating cavities painful.

A **cracked voice** in talking is due to an involuntary change from the chest to the falsetto voice as a result of the imperfect co-ordination of the movements of the vocal cords, and changes in the form of the larynx, trachea and other consonating cavities. It is noted especially in males at puberty, when the form of the larynx is changing; but it may persist throughout life.

Changes in the quality of the voice may result from any alterations in the cords and consonating cavities. These may be so extreme as to deprive it of every musical quality, and make it a mere noise for the enunciation of words. The nasal twang is observed when the pharyngeal and nasal cavities are not completely cut off in vocalization. When, in acute nasal catarrh, the passage of air through the nose is prevented, the condition known as "talking through the nose" is observed. When, in addition to the nasal obstruction, there is swelling of the soft palate and tonsils, the well-known guttural voice occurs. Changes in the walls of the chest, as in the emaciation of phthisis, produce the well-known hollow voice of that condition.

Stammering of the vocal cords is a term applied to a condition in which the patient is able to phonate ; but the act is accompanied by spasmodic contractions of the cords producing sudden interruption of the voice, but not interfering with articulation.

Diplophonia is a condition in which the pitch or qualities of the two vocal cords are not in unison. It is due to conditions which cause the vocal cords to vibrate differently, according to the degrees of tenseness or thickness of those structures, or to partial paralysis of one vocal cord, the other one being normal. It sometimes depends upon a division of the rima glottidis into anterior and posterior openings by small morbid growths, accumulations of mucus, or an irregular action of the muscles.

7. **Cough.**—Laryngeal disease may be accompanied by a variety of coughs, but the one generally regarded as characteristic is of brassy, barking quality, generally described as croupy, because most frequently observed in that disease.

Nearly as frequently observed is a dry cough, noticed in acute catarrhal laryngitis and in the early stages of phthisis. It is very harassing, and seems to be provoked by tickling sensations in the larynx.

A dry, hoarse cough is observed in cases of chronic laryngitis.

A cough with whoop or one in which the act of coughing is followed by a peculiar sound in inspiration occurs in whooping-cough. It is often followed by retching and vomiting.

Finally, we have the purely nervous cough, which may be either paroxysmal or continuous. It occurs in sexual neurasthenics, hypochondriacs, boys at puberty, and in those addicted to the excessive use of tobacco. It sometimes follows diphtheria, and occurs in the course of chlorosis. It is generally worse when the patient is under observation.

8. **Expectoration.**—The expectoration accompanying the great majority of cases of laryngeal disease is scanty in quantity, and clear and starchy as to quality. This statement applies with especial force to the catarrhal affections. When profuse in this class of cases, tracheal or bronchial involvement is suggested. Occasionally, purulent sputum is expelled from the larynx. This is observed in the destructive diseases, as perichondritis and all varieties of ulceration. When these are accompanied by necrosis of the laryngeal cartilages, the sputum may be very offensive in odor ; especially is this so in syphilitic and cancerous diseases.

Dry crusts of inspissated mucus may be expelled from the larynx in a condition which has been described as analogous to atrophic nasal catarrh, and has been called ozæna of the larynx.

Hæmorrhagic sputum is comparatively uncommon as proceeding from the larynx. Mucus streaked with small lines of blood is occasionally observed in acute laryngitis after violent paroxysms of coughing. In such cases small varicosities may be observed by the aid of the laryngoscope. The same symptom obtains in chronic laryngitis, in which affection expectoration of pure blood also may take place.

Blood changes, as in acute infections, purpura, leukæmia, chlorosis, and pernicious anæmia, are occasionally the cause of actual hæmorrhage from the larynx; but in these cases the hæmorrhage is nearly always sufficiently restricted in quantity to leave the primary disease as the most prominent factor. Laryngeal hæmorrhage may also result from hæmophilia, cirrhosis of the liver, and dilatation of the heart.

It is important, in studying laryngeal hæmorrhage clinically, to bear in mind that, like loss of blood from other localities, it is an accident having surgical aspects—brought about, it is true, by a mechanical change often slight, but rendered possible by previous alterations in the blood, or by degeneration or erosions of vessels.

Sometimes the blood escapes by diapedesis. Under such circumstances the seat of hæmorrhage may be any portion of the laryngeal cavity, although it is observed proceeding from either the ventricular bands or vocal cords. When due to erosion, it is observed most frequently at the posterior insertion of the cords, or on the anterior surface of the arytenoid cartilages.

Sometimes the hæmorrhage is submucous. As a rule, these extravasations are small; but they may be sufficiently large to produce blood tumors, which, in turn, excite obtrusive dyspnœa.

9. **Dyspnœa.**—Laryngeal dyspnœa is always due to obstruction, and this may be brought about by

a. Spasm.
b. Inflammatory swelling of the mucous membrane of the larynx. Pseudo-membranous formations in the larynx, as in diphtheria, a frequent and dangerous cause.
c. Œdematous swelling of the laryngeal tissues.
d. Tumors.
e. Foreign bodies.
f. Cicatricial contractures after syphilitic and lupoid ulcerations.
g. Paralysis of the adductors.
h. Paralysis of the abductors.
i. Disease of surrounding structures, which diminish the lumen of the larynx by pressure.

While laryngeal dyspnœa always proceeds from obstruction, it is hard to say what degree of stenosis is necessary to cause difficult breathing. High grades of dyspnœa have been observed when the patency of the larynx seemed hardly impaired, while respiration remained comparatively easy in what was apparently an almost complete stenosis. Nervous influences, circulatory conditions, inaccurate observations, and the idiosyncrasies of the patient, doubtless afford the explanation of anomalous facts.

The symptoms of laryngeal dyspnœa are characteristic, and serve to distinguish it clearly from that arising from other causes. It is in the majority of cases inspiratory as to time. A loud, stridulous sound is observed on inspiration; expiration is silent but prolonged. The larynx falls and rises with the acts of respiration. The head is thrown backwards, and the neck straightened. All soft parts about the trachea and thorax recede in inspiration. This is observed especially in the suprasternal notch and in the intercostal spaces, and by sinking in of the epigastrium. The patient is cyanotic, or has an ashy-gray color. He finally becomes unconscious from carbonic acid poisoning. He may be bathed in a cold perspiration. The respiration-rate is not increased; indeed, it becomes slower and slower as the condition progresses, until finally, before death, it consists of mere gasps. The heart-action is increased in frequency throughout.

Dysnpœa from tracheal disease is also obstructive, and consequently bears a certain resemblance to that originating in the larynx. The position of the patient is, however, different. The head is bent forward instead of backwards. The larynx does not move up and down with respiration, and the retraction of the soft parts on either side of the trachea is limited to the parts below the point of obstruction.

The acuteness of onset of laryngeal dyspnœa varies with the character of the pathological process. In œdema from Bright's disease or in angioneurotic œdema it is generally sudden, without preceding manifestations referable to the larynx. In laryngeal diphtheria it is more or less rapid. In secondary œdema, arising from already existing ulceration or from tumors, it will be preceded by disturbed respiration from the pre-existing lesion. The course of the disease may be disturbed by sudden exacerbations from time to time, from intercurrent accessions of infiltration.

Departures from the type of inspiratory dyspnœa are observed in some cases of laryngeal disease. Thus, in the case of a movable tumor in the subglottic space, the dyspnœa may be expiratory, as, during that period of respiration, the tumor is pushed upward and obstructs the glottis. Under such circumstances, emphysema is apt to develop. In laryngismus stridulus the act of inspiration is suddenly interrupted in its midst.

Information Obtained by Examination of the Larynx Externally.

An examination of the larynx externally affords but little information. It is true that there is a slight tenderness observed in some cases of catarrhal inflammation, and in lupus this may amount to soreness. In perichondritis the sensitiveness may amount to actual pain. In the latter condition, when the disintegration of tissue is advanced, there may be felt undoubted crepitus. Swelling or enlargement of the larynx may be observed in cases of perichondritis and tumors, especially in carcinoma. The external movements of the larynx are increased in dyspnœa from laryngeal stenosis.

Constitutional Symptoms of Laryngeal Disease.

These are very few. Fever of mild degree accompanies the early stages of acute catarrhal inflammation. Cyanosis accompanies stenosis. Diphtheria and malignant disease are associated with the usual general manifestations of those conditions occurring in other parts of the body. Owing to the anatomical construction of the larynx, the pathological process is well confined, and is not so apt to affect the general system as when located elsewhere.

Synopsis of Symptoms and Clinical Course of the Various Diseases of the Larynx.

1. Acute Catarrhal Laryngitis

Is due to exposure to cold, especially in those who are predisposed by reason of chronic nasal and nasopharyngeal disease. Bosworth takes the extreme ground that exposure is inoperative as a cause unless there is preexistent disease of the nose and pharynx. In the majority of cases in those who have regarded themselves as in perfect health, the laryngeal catarrh is preceded by similar changes in the nasal cavities. The symptoms of acute catarrhal laryngitis are of rapid onset, consisting of slight fever, aphonia more or less marked, sensations of rawness, tickling or "stuffiness" in the larynx, dry cough in the earliest stage, later a cough somewhat moist, with scanty but clear expectoration. On laryngoscopic examination, the mucous membrane of the larynx is found to exhibit the bright redness characteristic of acute catarrhal inflammations in all localities. The cords themselves are injected.

2. Acute Catarrhal Laryngitis in Children

Is brought about by exposure to cold, especially in those predisposed by lymphatism. Two varieties have been described, viz.: *Acute supraglottic laryngitis* and *acute subglottic laryngitis.*

In acute supraglottic laryngitis, fever is mild or absent; the voice is hoarse or metallic. Some tenderness is evident on external pressure over the larynx. The disease runs a course of five to ten days.

In acute subglottic laryngitis, the fever is moderate or high. Hoarseness is prominent and of rapid onset; it may amount to absolute aphonia.

The cough is harsh and barking (the so-called croupy cough), and later is attended by some expectoration. Inspiration is often crowing from subglottic obstruction. The symptoms are greatly exaggerated at night. The laryngoscope shows prominent bulging, with inflammation of the mucous membrane of the larynx below the vocal cords.

3. Chronic Catarrhal Laryngitis

Is a sequence of disease of the higher air-passages in nearly every instance. Its incidence is probably influenced by alcohol, tobacco, indigestion, inhalation of dust, and improper use of the voice. The onset of the symptoms is slow. The patient first complains of a husky voice, this condition being most pronounced in the morning, but disappears in a measure on clearing the throat or by use of the voice. The voice grows weaker during the day. The laryngoscope shows the mucous membrane of the larynx to be hyperæmic and swollen; the vocal cords are grayish and present an irregular surface. The disease runs an indefinite course. It is very often associated with bronchitis or tracheitis.

4. Chronic Subglottic Laryngitis

Is commonly the result of lymphatism. Its onset is insidious. There is slow but progressive impairment of the voice, which may finally amount to complete aphonia. The cough is prominent and distressing in the fully-developed disorder. It may be harsh, metallic and croupy. There may be inspiratory and expiratory dyspnœa, with stridor. With the laryngoscope the subglottic mucous membrane is seen to project from beneath the vocal cords as two rounded or oval-shaped masses, which may be grayish and opaque or of a deep inflammatory tinge. The disease is incurable. Sooner or later tracheotomy, with permanent wearing of the tube, will be found necessary.

5. Chorditis Tuberosa or Singers' Nodes

Is a condition caused by the sustaining of high notes by singers who have chronic laryngitis. The disease is really but a variety of chronic laryngitis. It is characterized by the appearance of a nodule on one or both vocal cords midway between the vocal processes and the anterior insertion of the cords. The lesions exhibit but little tendency to progress after development. The symptoms appealing to the patient include hoarseness and inability to sing the high notes.

6. Laryngitis Sicca

Is practically always associated with atrophic rhinitis or pharyngitis. The patient complains of a more or less complete aphonia with dyspnœa on awakening in the morning, and relieved by clearing the throat, greenish-yellow masses or crusts being expelled. The breath is fœtid during oral respiration. This makes a distinction between laryngitis sicca and atrophic rhinitis. In the latter, the fœtid odor is detected during nasal respiration. Examination with the laryngoscope discovers the presence of greenish-gray masses adhering to the subglottic mucous membrane. The disease pursues a chronic course; it is incurable, but may be relieved by treatment.

7. Acute Phlegmonous Laryngitis

Is probably a true erysipelas of the larynx. While commonly attributed to exposure to cold, it is inconceivable that such an inflammation is possible without infection. Its symptoms are of rapid onset, and include chill, followed promptly by fever, dyspnœa, aphonia, and stridulous breathing, developing rapidly unless tracheotomy is performed. It is apt to terminate fatally. The less severe cases end in resolution or in formation of abscess. The mucous membrane of the larynx is found to be bright-red, tense, and semi-opaque. The epiglottis and the ventricular bands are swollen, the glottic chink being triangular. The disease reaches its height in about three or four days.

8. Œdema of the Larynx

May be brought about by renal disease, angio-neurotic œdema, pressure upon blood-vessels, or various local disorders. The patient exhibits inspiratory dyspnœa, with stridulous respiration and loss of voice, usually coming on suddenly. There is a sense of fullness and distention in the larynx. The patient is often cyanosed. Examination shows œdema of the epiglottis and distention of the aryepiglottic folds, the glottic chink being triangular. The disease runs a rapid course of from two or three to thirty-six hours. As a rule, treatment is required to check its course.

9. Membranous Laryngitis; Membranous Croup.

Notwithstanding the advances made in our knowledge of laryngeal diseases, opinions respecting the unity of membranous croup and diphtheria are as divergent as ever, though the trend of teaching is in the direction of regarding the two conditions as identical. From a practical standpoint, the teachings of Goodno approach as near the truth as we can at present go. He says: "It seems probable, however, that, despite the doctrine of Strumpell that 'the proposition that there are two distinct diseases, croup and diphtheria, is absolutely untenable,' a membranous inflammation of the larynx not due to the action of the germs of diphtheria *may* exist. Inasmuch, however, as many of our most eminent clinicians consider its differentiation difficult or impossible, it is incumbent upon practitioners to regard all cases of laryngitis marked by membranous exudate as infectious, and thereby avoid the risk of communicating the disease to others. The various pathological and clinical differences which have been contended for have not yet been established, and it is therefore proper to consider the subject under one heading."

In those cases in which the laryngeal symptoms appear subsequently to the presence of diphtheritic manifestations in the pharynx, the nature of the case is beyond all question. We do not hesitate to call such instances examples of laryngeal diphtheria. The diagnosis of the primary laryngeal cases is always a difficult matter. In the majority of such surviving for a sufficient length of time, the diphtheritic membrane appears in the pharynx, and then the diagnosis is clearly defined.

The clinical manifestations of membranous laryngitis are, at first, identical with those of the catarrhal variety. The patient exhibits a mild degree of fever, with cough and hoarseness which suggest the larynx as the *locus mali*. But in a very short time the symptoms of laryngeal obstruction are added. At first these are paroxysmal, or, at most, appear mainly at night, each night presenting a distinct advance in severity over the same period preceding. Finally, the obstructed respiration becomes continuous. Inspiration becomes more difficult than expiration. Respiration is stridulous. The little sufferer becomes cyanotic, and then unconscious. There may be expectoration of membranous formations, and this settles the nature of the pathological process. The prognosis of laryngeal diphtheria is most unfavorable.

10. Laryngeal Tuberculosis or Tubercular Laryngitis

Is generally secondary to tubercular processes elsewhere; but too much dependence must not be placed upon this statement, for tubercular laryngitis may be a primary affection. It is first manifested by hoarseness, which proceeds to a more or less complete aphonia, associated with pain, dysphagia, cough, and dyspnœa. The relative severity of these symptoms depends upon the situation of the lesions. Pallor of the laryngeal and pharyngeal mucous membrane usually precedes the characteristic tubercular changes. The epiglottis is often enlarged, appearing as a pale, turbanlike body. The arytenoid cartilages are pale, puffy, and pyriform. Later there appear small superficial ulcers, which coalesce, and cause the parts to present a "worm-eaten" appearance. Next there are infiltrations and ulcerations, beginning on one or both vocal cords. In the later stages of the disease the mucous membrane of the larynx becomes one mass of ulcerations, which may extend in depth, destroying the cartilages. (*Vide* p. 345 for tabular differentiation of laryngeal tuberculosis and laryngeal syphilis.)

11. Laryngeal Lupus; Lupoid Ulceration of the Larynx

Is due to unknown causes. It is practically always associated with manifestations of lupus on the skin and in the pharynx. It runs a very chronic course. It begins as nodules ranging in size from that of a pin-head to that of a split pea, and greatly deforming the parts. These finally become softer, or "apple-jelly like," in appearance, and ulcerate. The ulceration progresses slowly, healing in one direction while it advances in another. It generally attacks the free edge of the epiglottis first. Pain is practically always absent. Hoarseness and aphonia are observed when the vocal cords are involved. In advanced cases, cicatricial stenosis with dyspnœa occurs. Destruction of the cartilages is rare. In doubtful cases, lupus may not be certainly differentiated from syphilis until the therapeutic test has been made.

12. Laryngeal Syphilis

May be manifested as simple catarrh, mucous patches, condylomata, ulceration, diffuse infiltration, fibroid metamorphosis, cicatrices, perichondritis, and paralyses. All of these lesions, with the exception of diffuse infiltration and fibroid metamorphosis, have been described sufficiently in the pages devoted to laryngeal symptomatology.

Diffuse infiltration attacks the epiglottis, vocal cords, and interarytenoid folds. It produces considerable distortion, aphonia, and dyspnœa. The condition is due to small-celled infiltration, and this may become organized into connective tissue. Repeated attacks are associated with fibroid metamorphosis, leading to increased and increasing deformity, and even great stenosis. These cases are very resistant to treatment, if not actually incurable.

13. Laryngeal Perichondritis

May be either primary or secondary. The primary cases are due to exposure to cold ; the secondary to syphilis, tubercular or malignant ulceration, acute infectious diseases, gout, and traumatism. The acute form is ushered in with sudden chill, fever, dyspnœa, tumefaction of the cartilages, hoarseness, etc. The symptoms, of course, are modified by the nature of the primary affection. When there is suppuration with necrosis of the cartilages, there are, as additional signs, crepitus, purulent discharge, fœtor, cicatricial contraction and stenosis, etc. The outlook for the restoration of the voice and the lumen of the larynx is unfavorable.

14. Benign Growths of the Larynx

Produce loss of voice, dyspnœa, slight cough. When the epiglottis is involved, there is dysphagia. (*Vide* p. 346.)

15. Malignant Growths of the Larynx

Find their causes in heredity, prolonged local irritation, middle life and beyond, and the male sex. The objective appearances have already been described. The symptoms are of gradual onset and include hoarseness, pain, cough, and tenderness on pressure. There are tenacious purulent discharges, fœtor, dysphagia when the growth involves the epiglottis, and hæmorrhages, which are usually slight in the early stages and severe in advanced cases. The cancerous cachexia is usually not manifested until the growth extends to the pharynx. A fatal issue is to be expected within, at the most, three years.

CHAPTER XI.

PHYSICAL EXAMINATION OF THE RESPIRATORY ORGANS.

THE methods to be employed for the physical examination of the chest include

1. Inspection.
2. Mensuration.
3. Palpation.
4. Percussion.
5. Auscultation.

Valuable as these are in establishing a diagnosis, the physician should not rely upon them to the exclusion of data otherwise obtained, but should base his conclusions upon both objective and subjective phenomena and the history of the case.

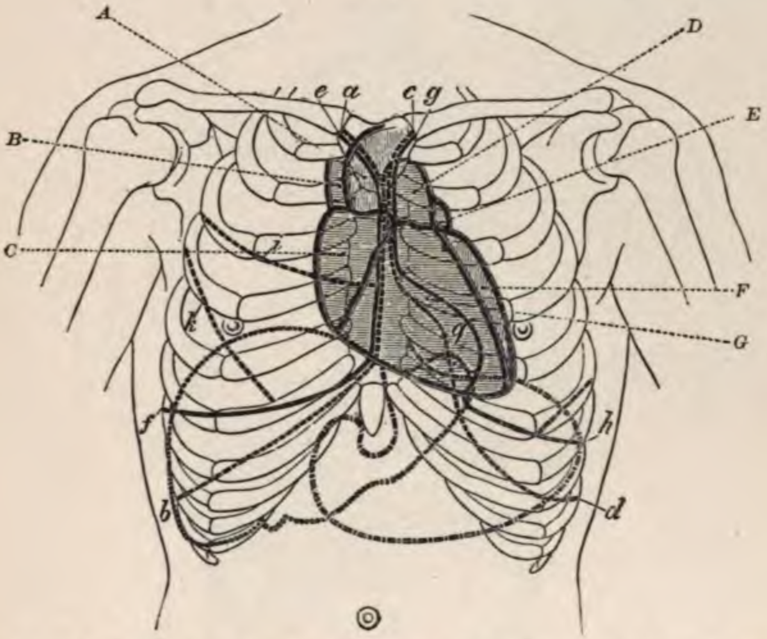
1. **Inspection.**—Accuracy of results from inspection of the chest is to be obtained only by attention to detail and giving one's self every opportunity for gaining full information. To this end, the chest should be perfectly bare. The patient must stand or sit erect, with arms hanging symmetrically by the sides. Illumination must be good. While, under ordinary circumstances, direct lighting is all that is required, the best results may be obtained only when the parts are inspected, also, with the light coming from the side. In the latter case, depressions are made more evident, and slight irregularities become more noticeable. The examination should be conducted with attention to the following points:

a. The size and shape of the chest, observing particularly whether there exist
   i. General alterations in the shape of the chest.
   ii. Unilateral alterations.
   iii. Local changes, as bulgings, and depressions.
b. The respiratory movements.

The Normal Chest.—The shape and size of the normal chest varies according to the age of the subject. In infants and young children, the antero-posterior and transverse diameters are about equal, and the contour of the chest a nearly perfect circle. After the second year, changes approaching that of the adult chest gradually take place, the transverse diameter becoming the greater, and the outline of the chest assumes the shape of an ellipse. With the advent of old age, the circular shape of infancy is resumed in part.

A proper conception of the shape of the normal chest is to be obtained only by experience gained from repeated observations. This gained, the trained eye recognizes immediately all departures from the normal. Attention should be paid to the position of the sternum and its general relations. Normally, it projects forwards from above downwards, and is slightly more prominent than the balance of the thoracic walls. Slight depressions or prominence at the ensiform cartilage may be disregarded.

FIG. 59.



Anterior surface of chest. ——, continuous line, border of lungs; ----, broken line, fissures between lobes and line pleura; ......, dotted line, liver; -·-·-·-, dot and dash line, stomach; ═══, double line, heart and great vessels. *A*, Aorta; *B*, vena cava superior; *C*, right auricle; *D*, pulmonary artery; *E*, left auricle; *F*, left ventricle; *G*, right ventricle; *ab*, border of right pleural sac; *cd*, border of left pleural sac; *ef*, edge of right lung; *gh*, edge of left lung; *i*, fissure between right upper and middle lobes; *k*, fissure between right middle and lower lobes. (From Sahli's "Lehrbuch der klinischen Untersuchungs. Methoden," etc.)

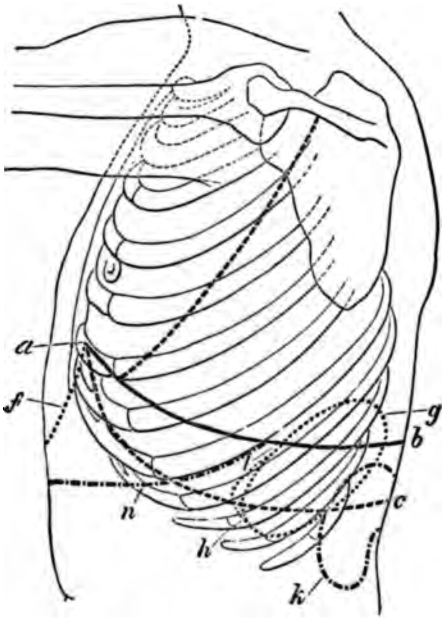
Aside from a slight enlargement discoverable only by mensuration, the two sides of the chest are perfectly symmetrical. If the general nutrition of the patient is fair or good, there will be no depression in the infra- and supra-clavicular spaces.

Various types of abnormally-shaped chests have been described. Not much information of diagnostic value is suggested by *size of the chest*, as departures from the normal standard in this respect are perfectly consistent with good health. The chest is abnormally small in all persons who have been long ill from any cause. It may also result from rachitis and post-nasal adenoids in childhood. A very large chest is observed in connection with emphysema.

General Alterations in the Shape of the Chest.—These include:

a. The alar or pterygoid chest.
b. The flat chest.
c. The rachitic chest.
d. Bilateral diminution in size.
e. Bilateral enlargement.

FIG. 60.



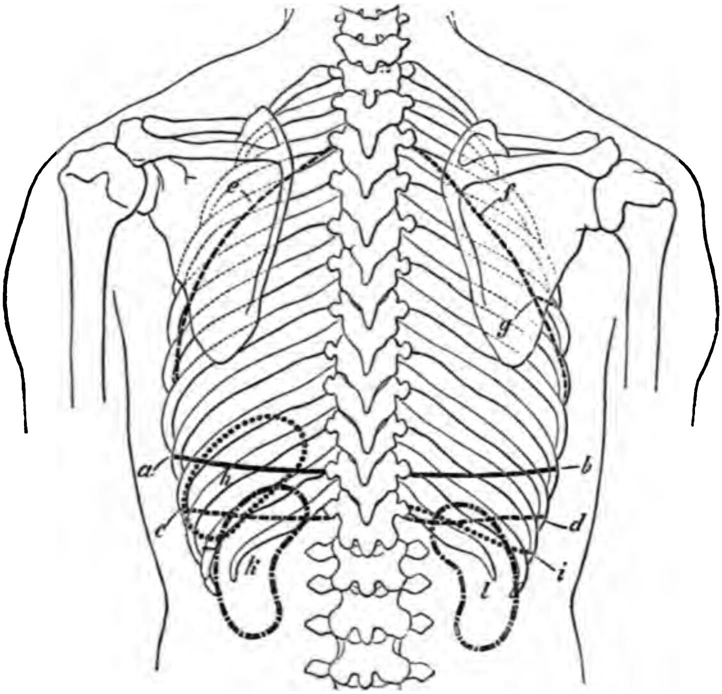
Lateral surface of chest. ——, continuous line, lower border of left lung; - - - -, broken line, fissure between left upper and lower lobes, lower border of pleura; ......, dotted line, spleen and liver; ·-·-·-, dot and dash line, stomach and kidneys; *ab*, lower edge of left lung; *ac*, lower border of pleural sac; *f*, edge of left lobe of liver; *g*, posterior; *h*, anterior end of spleen (when oval in shape); *k*, lower edge of left kidney; *n*, greater curvature of stomach.—After Sahli.

(*a*) *The Alar or Pterygoid Chest.*—This type of chest has also been designated "*the paralytic thorax.*" It is characterized by flattening of the anterior surface of the chest, by depressions in the supra- and infra-clavicular fossæ, by stooping shoulders, unusual projection of the scapular angles, and the angle at which the ribs join the sternum is unnaturally acute. This type of chest may occur in persons whose respiratory organs are perfectly sound. Pathologically, it is observed in those with phthisis, or with a predisposition to that disease.

(*b*) *The Flat Chest.*—In this type, the flattening takes place at the expense of the antero-posterior diameter. The sternum is depressed below the level of the costal cartilages, and the angle at which the true ribs join the sternum is near a right angle. The flat chest indicates deficient respiratory capacity.

(*c*) *The Rachitic Chest.*—A variety of general deformities of the chest may result from rachitis. Most characteristic of these is the "***pigeon-breast***," in which condition the sternum is unduly prominent, the deformity being made all the more evident by a slight grooving of the costal cartilages on either side. The lateral walls of the thorax are flattened, so that a transverse section of the chest is more or less triangular.

FIG. 61.



Posterior surface of chest. ——, continuous line, lower border of lungs; ----, broken line, fissures between lobes and line of pleura; ......, dotted line, spleen and liver; ·-·-·-, dot and dash line, kidneys; *e*, fissure between left upper and lower lobes; *f*, fissure between right upper and lower lobes; *g*, beginning of fissure between right upper and middle lobes; *ab*, lower edge of lung; *cd*, lower border of pleural sac; *h*, spleen; *i*, lower border of liver; *k*, left kidney; *l*, right kidney.—After Sahli.

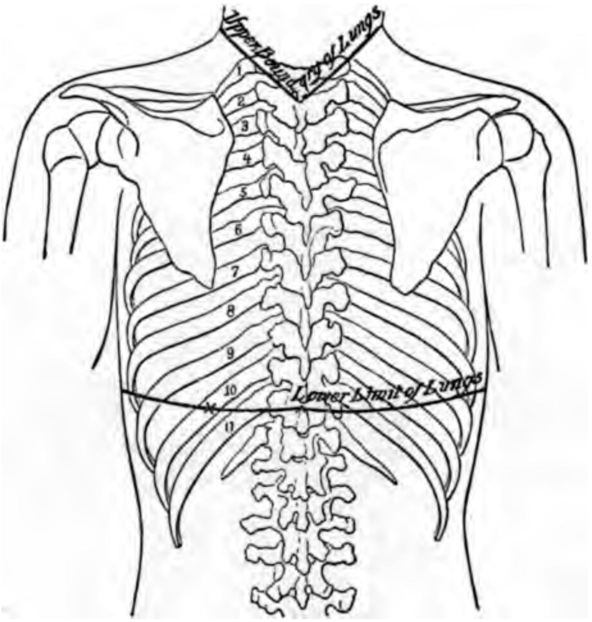
The "*funnel-breast*" and "*Harrison's sulcus*" also occur in rachitis.

The type of rachitic chest of which much is said is seen only in young children. Reference is here made to the so-called "*beading of the ribs*," or "***rachitic rosary***." It is characterized by a row of prominences discoverable usually by palpation only, though sometimes also by inspection, along the line of the junction of the ribs with their respective cartilages.

In still another type the lower ribs flare out, owing to the pressure of an enlarged liver beneath, the depression above being due to diaphragmatic contractions.

(*d*) *Bilateral Diminution in Size of Chest.*—This condition indicates diminished respiratory capacity. It is usually the result of atrophic conditions of the lungs, as from old age. The lower ribs are more obliquely placed, and the diaphragm higher than in a normal chest. There is bilateral diminution in size in the deformity already mentioned as the pterygoid or alar chest, and in the chest of pulmonary tuberculosis.

FIG. 62.



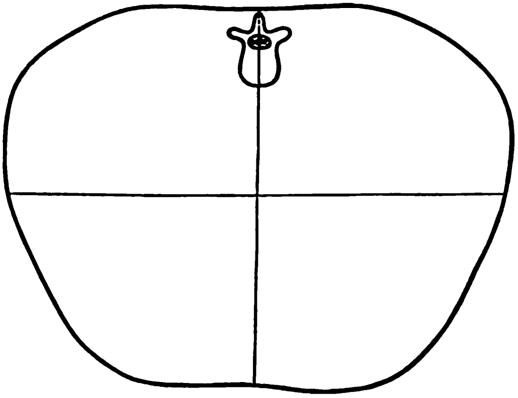
Posterior surface of chest, showing the relation of the upper and lower limits of the lungs to the chest-wall.—After Sahli.

(*e*) *Bilateral Enlargement.*—In this type of chest all the diameters are increased, especially the antero-posterior. A transverse section of the thorax is approximately circular. The sternal curve forward is especially prominent. The back and shoulders are rounded. The ribs are almost horizontal, and this widens the angle of the costal arches at the epigastrium.

The respiratory capacity of a chest of this type is greatly impaired, because of the defective movements during respiration. The position of the chest being that of full inspiration, but little additional expansion can be accomplished. Expiration is difficult. The difference in circumference between full inspiration and complete expiration does not amount to much more than an inch or an inch and a half. To atone for the restricted respiratory movements, increased frequency is necessary, inspiration being short, and expiration prolonged. There is more or less dyspnœa, and this is evidenced by the drawn and anxious face, and the livid or purple lips.

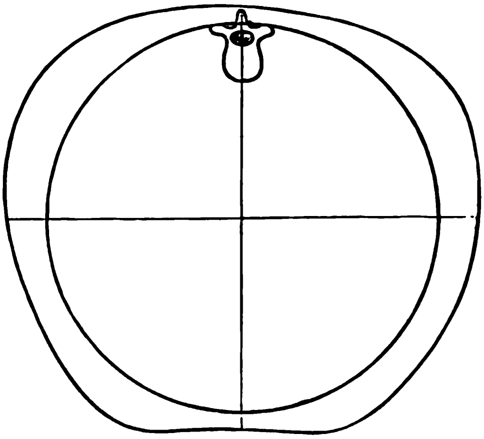
Bilateral enlargement of the chest indicates enlargement of the thoracic contents. Usually it is the result of pulmonary emphysema. Theoretically, it may be produced by double pleural effusion; but the latter condition, when sufficiently great to effect this deformity, is almost incompatible with life.

FIG. 63.



Transverse section of healthy adult chest upon level of sterno-xiphoid articulation.—After Gee.

FIG. 64.

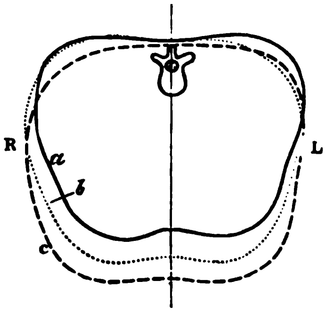


Transverse section of chest of an infant aged 9 months. A circle drawn within the tracing for the sake of comparison.—After Gee.

Hence, emphysema remains, from a practical standpoint, the sole cause. Much confusion respecting the chest of emphysema has arisen by reason of the application to it of the term "*barrel chest.*" As a matter of fact, the cases in which such a term is applicable are rarely, if ever, seen. Accuracy of teaching demands, therefore, that the term "barrel chest" be dropped from our nomenclature.

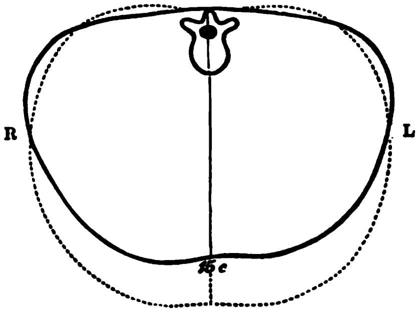
ii. **Unilateral Alterations in Size.**—An *increase in the size of one lateral half of the chest* indicates increased contents of that side, and is especially suggestive of unilateral pleural effusion. Exceptionally, it may result from emphysema, the pathological process spending its force on one lung. It also occurs in pneumothorax and intra-thoracic tumors, hæmothorax, compensatory hypertrophy of one lung, the other lung being extensively diseased by some chronic process. Unilateral enlargement of the chest is readily recognized by inspection. Actual measurement usually demonstrates that the difference in the two sides is not so great as one would expect. The affected side assumes a circular form, owing to increase in the antero-posterior diameter. In the case of pleural effusion and pneumothorax, the soft parts of the intercostal spaces and depressions between the ribs are obliterated. Actual prominence of the intercostal spaces is rarely, if ever, seen. In the majority of cases of one-sided pleural

FIG. 65.



Horizontal section of chest of a child 2 years old. a = chest at rest; b = chest after fullest possible expansion; c = chest after forcible injection of air into both pleural cavities.

FIG. 66.

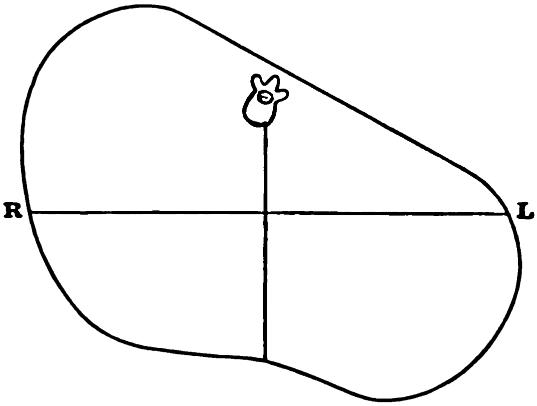


Alar thorax in a child. Dotted line indicates the normal.—After Gee.

effusion both halves of the chest are enlarged, the healthy side being expanded to a slight degree because the pressure on the diseased side exerts some influence on the entire thoracic contents.

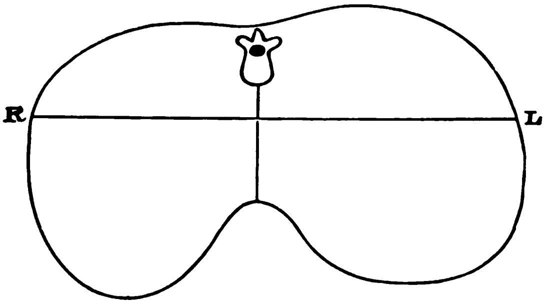
Unilateral decrease in the size of the chest may be observed in pulmonary tuberculosis, especially in the fibroid cases; after the absorption of a pleuritic effusion, the lung not being in a condition to resume its former shape; and in obstruction of a main bronchus. The deformity is, as a rule, especially noticeable below the clavicle or in the axilla, or in both situations. Its recognition by inspection is made easier by its usual association with a compensatory hypertrophy of the opposite lung.

FIG. 67.



Shape of chest in scoliosis.—After Gee.

FIG. 68.

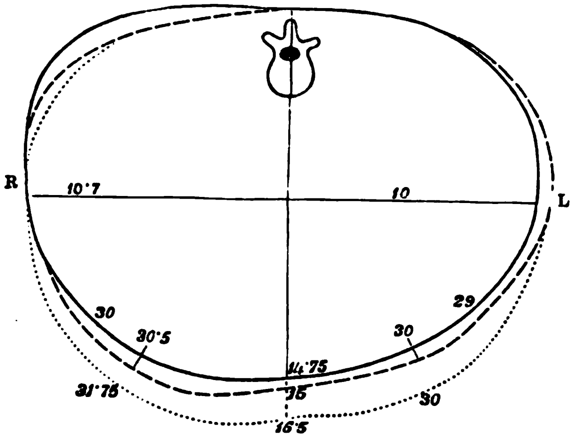


Cup-shaped depression.—After Gee.

iii. **Local Changes, as Bulgings or Depressions.**—Most of the local bulgings of the thoracic walls have to do with other than respiratory diseases, and these will receive attention in a subsequent chapter. In this place we need mention only the possibility of the condition arising from a localized pleural effusion, a pointing empyema, or a hernia of the lung.

The most important of the *localized depressions* are those observed in the supra- and infra-clavicular regions in pulmonary tuberculosis as the result of shrinking of the apices of the lungs.

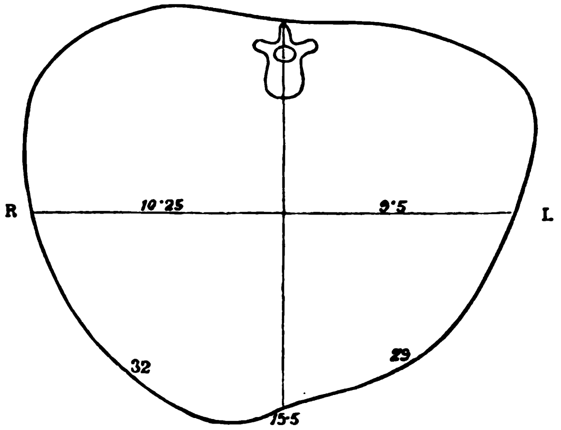
FIG. 69.



Unilateral enlargement of chest (right side); artificially produced by injecting air into the right pleural cavity. Unbroken line = outline before injection. Broken line = outline after moderate distention. Dotted line = outline after extreme distention. Figures at bottom of vertical line indicate the antero-posterior diameters; along horizontal line, indicate transverse semi-diameters. Remaining figures indicate right and left semi-circumferences.—After Gee.

A deep depression at the lower end of the sternum constitutes the deformity known as "*funnel-breast*," or "*trichterbrust*" of the Germans.

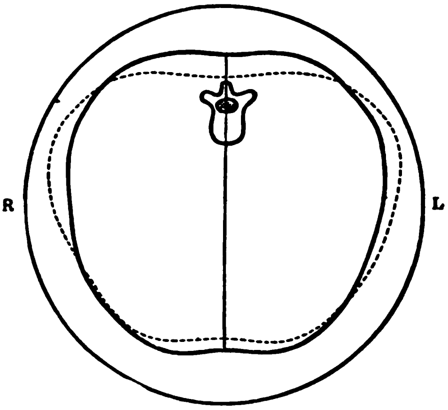
FIG. 70.



Unilateral retraction of chest consequent upon cirrhosis of left lung in a girl of fourteen. Figures as in preceding sketch.—After Gee.

It is usually a congenital condition, though it may be acquired by those whose occupations force them to maintain more or less constant pressure in this locality, as in the case of shoemakers. Its presence is not inconsistent with integrity of the pulmonary functions. It is generally regarded, however, as a stigma of degeneracy.

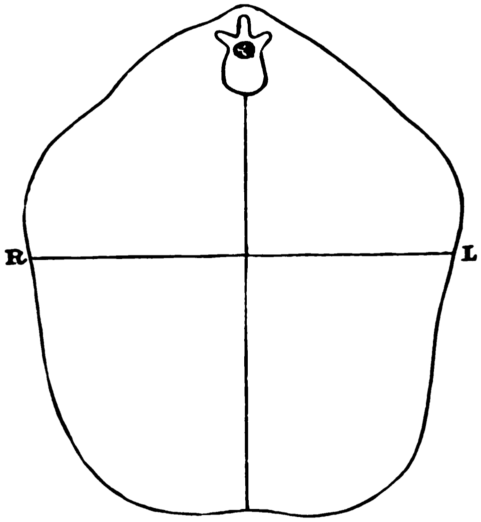
FIG. 71.



Bilateral enlargement of empyema. Inner line = emphysematous chest. Outer line = a circle drawn to show how nearly the emphysematous chest approaches the circular shape. Dotted line = natural adult chest.—After Gee.

The thorax *en gouttière* possesses about the same clinical significance as the funnel-breast. It is characterized by a trough-like depression, the

FIG. 72.



Shape of chest in angular curvature. Tracing taken from a man aged 52 years.—After Gee.

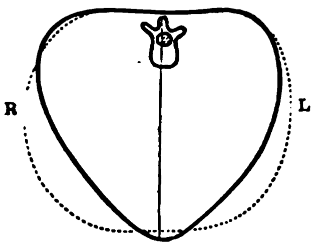
bottom of which is formed by the sternum, and its sides by the incurved ribs.

(*b*) **The Respiratory Movements.**—Much that has an important bearing on this department of physical examination of the chest has already been reviewed in the chapter on "Respiration," and will be mentioned at this time only as incidental to additional data. When observing the movements of the chest and noting any alterations in them, attention should be directed to the character of the alterations, and the portions of the chest which may participate in them. These alterations include the following:

- i. Bilateral alterations in movements.
  - *i.* Increased frequency.
  - *ii.* Decreased frequency.
- ii. Unilateral changes of movement.
  - *i.* Increased movement of one side.
  - *ii.* Decreased movement of one side.

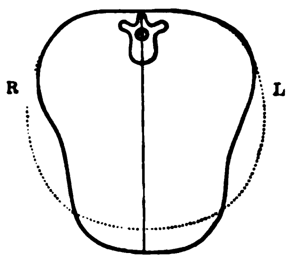
i. **Bilateral Alterations in Movements.**—The conditions characterized by *increased respiratory movements* include all acute lung diseases, especially those attended by fever, fevers generally, and in numerous other conditions to which reference has been made when speaking of respiratory symptoms (p. 121).

FIG. 73.



Pigeon-Breast. Tracing taken from a child of 7 years. Dotted line indicates natural shape at same age.—After Gee.

FIG. 74.



Rickety chest. Dotted line indicates shape of chest in healthy infant of about the same age. —After Gee.

Decreased frequency of movement is never of pulmonary origin. It results from lesions of the respiratory centre in the medulla and from the action on the same of certain poisons, as opium and in uræmia.

Actual decrease in the extent of the movements occurs from

- *i.* Deficient entrance of air into the lungs.
- *ii.* Collapse or consolidation of the lungs.
- *iii.* Distention of the air-cells, as in emphysema.
- *iv.* Weakness or paralysis of the respiratory muscles.

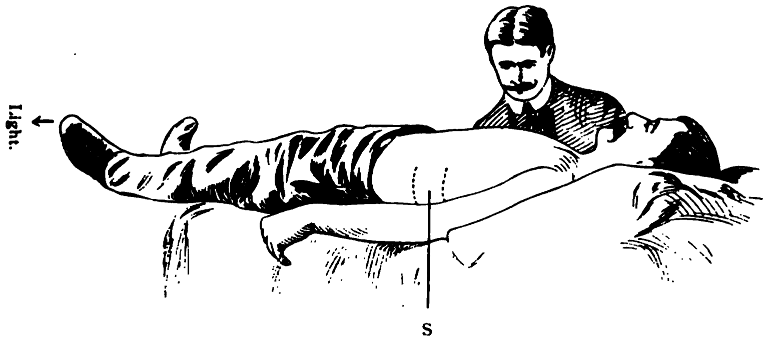
ii. **Unilateral Increase of Chest Movements.**—This condition may be observed in any case in which the functional activity of one lung is greatly impaired, the other side compensating for the deficiency by taking on increased action. With this, there is generally enlargement of the active side, with hyper-resonance on percussion.

Unilateral Decrease of Movements.—This occurs in all conditions characterized by lesions which lessen the area of respiratory surface. Hence it may be observed in croupous pneumonia. It is also symptomatic in the occlusion of a bronchus, unilateral pleurisy with effusion, pneumothorax, retraction of one lung, pulmonary adhesions, and occasionally from pressure from below the diaphragm, as in enlargements and tumors of the liver or spleen.

It is not uncommonly stated that the movements of one side of the chest are impaired in cases of hemiplegia. Such, however, is unlikely to occur, because the respiratory muscles have bilateral representation in the brain, and hence are liable to escape the serious loss of power which affects the voluntary muscles.

Litten's Diaphragmatic Shadow.—To obtain this phenomenon, attention to detail in the placing of the patient and the direction of the light is absolutely necessary. The patient is placed in a recumbent position upon his back, with his chest perfectly bare. The light must come from the direction of his feet; all other sources of illumination must be positively excluded. The physician should stand at the side of the patient. Now the latter is directed to take a long breath. As he does so, a shadow, starting at about the seventh rib, moves along the axillary line to the ninth or tenth rib. With full expiration, the shadow moves upwards to its former position. Litten's shadow phenomenon is never absent in health, unless it be in unusually corpulent individuals, and is best seen in spare, muscular subjects. The average normal excursion of the shadow movements is about two and a half inches. With forced breathing it may be increased to three and a half inches.

FIG. 75.



Position of physician and patient for eliciting Litten's shadow. S indicates position of the shadow.

The moving shadow, as above described, is due to the separation of the diaphragm from the inner walls of the thorax and the lowering of its dome during deep inspiration, and the filling-in of the space thus vacated by normal lung-tissue.

The diaphragmatic shadow is absent in pneumonia of the lower lobe, extensive pleuritic adhesions, and advanced pulmonary emphysema. The movement is limited in range in cases of early phthisis.

Abdominal tumors and effusions, unless large, do not affect it to any great extent.

The extent of the shadowy movement is generally regarded as a fair index of the respiratory capacity of the patient.

2. **Palpation.**—Palpation confirms the information obtained by inspection, and, in addition, gives information as to

a. The condition of the vocal fremitus.
b. The presence of bronchial or rhonchal fremitus.
c. The presence or absence of friction fremitus.
d. Undue resistance of the soft parts.
e. Sense of fluctuation in the intercostal spaces.

In the practice of palpation, the observer should stand on the side of the patient opposite to the surface he is examining. The parts must be bared. The hands, well warmed, should then be placed upon corresponding points on each side of the chest, and observations made.

(*a*) **The Vocal Fremitus.**—The vocal fremitus consists of vibrations imparted to the walls of the chest through the columns of air in the bronchial tubes by the act of speaking. In health it is modified by the pitch of the voice, being more noticeable in those with a bass intonation; it is more pronounced in male subjects, and in those with thin chest-walls; it is more marked over the right apex than over the left, because the right bronchus is larger, and its angle with the trachea more acute. It is scarcely perceptible over the scapulæ behind and the præcordia in front. The sound to be utilized for eliciting it should be uttered in a monotone, and consist of some standard vocal exercise, as counting one, two, three, or repeatedly uttering the words ninety-nine.

As the result of disease, the vocal fremitus may be (i) increased or (ii) decreased or absent.

(i) **Vocal Fremitus Increased.**—Any morbid condition which favors the conduction of sound-vibrations increases the vocal fremitus. Thus we find it unduly prominent over all pulmonary consolidations, as in pneumonia and tuberculosis.

(ii) **Vocal Fremitus Diminished or Absent.**—Conversely, the vocal fremitus is diminished by any causes which interfere with the conduction of sound-vibrations. These include slight pleural effusion, thickening of the pleura and pulmonary emphysema, cavities filled with fluid, and partial occlusion of a bronchus.

It is absent when a bronchus is entirely obstructed, in marked pleural effusion, in pneumothorax, pyothorax, and hæmothorax.

(*b*) **The Bronchial or Rhonchal Fremitus.**—The bronchial fremitus consists of vibrations imparted to the walls of the chest by the passage of air through or over the accumulations of mucus or other fluid in the bronchial tubes. They vary greatly in their rhythm. They are usually general, and are found in asthma, bronchitis (especially in children), and in œdema of the lungs. They may be localized over a cavity containing fluid. Bronchial fremitus is sometimes referred to as "palpable râles."

(*c*) **Friction Fremitus.**—Friction fremitus is very rarely observed. It is due to the rubbing of opposing pleural surfaces which have been roughened by inflammation. It is usually most pronounced when present at the base of the axillary region, where the diaphragmatic pleura comes in contact with the costal pleura. When present, its diagnostic significance is undoubted, as it indicates pleurisy before the stage of effusion (dry pleurisy).

(*d*) **Undue Resistance of the Soft Parts.**—When from any cause the intrathoracic contents are increased, as in pleural effusion, palpation discovers increased resistance.

(*e*) **Sense of fluctuation in the intercostal spaces** may be observed in pleurisy with effusion or in pointing empyema.

3. **Percussion.**—The practice of percussion in the study of disease has been greatly hampered by false ideas concerning the acquirement of the art. On the one hand is the belief that it is a most difficult subject, which few clinicians ever master; and, on the other, the notion that its principles may be acquired automatically or intuitively. If, however, the study and practice of percussion are approached in the same spirit as in any other branch of medicine, that is, with the determination first to master the fundamental principles and then apply them systematically in the investigation of disease, the subject will present but little difficulty. It is necessary at the start that the student acquire proper conceptions as to what constitutes a normal chest. Unless he has done this, he can never determine with certainty what is abnormal. The late Austin Flint is said to have taught that no student of medicine should be permitted to examine a diseased chest until he had studied most carefully at least fifty normal ones. Dry as such preparation may prove to be, it is certain that the time thus spent is never wasted, and will save much labor and anxiety in after years. So we say:

A prerequisite for accuracy in the physical examination of the chest is a conception of the physical signs, and their variations, as observed in health.

It is also important that the student acquire a good technique in the practice of percussion. Indeed, it is necessary that he should learn the details so perfectly, and practice them so repeatedly, that the mechanical part of the percussion becomes automatic, so that the mind may be directed exclusively to the interpretation of the sounds produced.

Percussion may be practised in two ways: *Immediate percussion*, in which the blow is applied directly to the chest, and *mediate percussion*, in which the blow is applied to the finger or an instrument over the part to be examined. The latter is practically the only method now employed.

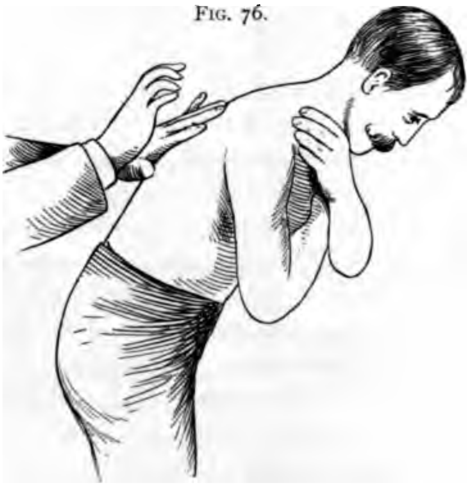
Technique of Percussion.—Proper practice of percussion involves attention to the following details:

a. The position of the patient.
b. The manner of performing percussion.
c. The interpretation of the sounds elicited.

(*a*) **The Position of the Patient.**—The patient may be either sitting or lying. He must be placed symmetrically and thoroughly relaxed. It is necessary that his back be well supported, because the pressure of the finger placed against the chest may disturb his equilibrium if this is neglected.

When percussing the back, the patient should be instructed to sit or stand with his body bent forwards, and his arms crossed, his hands resting upon opposite shoulders. In this position, the scapulæ are gotten as much out of the way as is possible. The stooped position also favors the application of the finger as the pleximeter.

FIG. 76.



Position of patient for percussion of back.

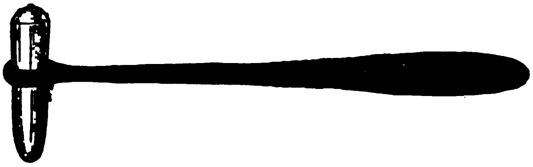
When percussing the axillary regions, the arms should be elevated, and bent over the top of the head.

(*b*) **The Manner of Performing Percussion.**—As already stated, mediate percussion gives so much better results than the immediate that it is practically the only method employed in medical work. The instrument against which the blow is applied is called a *pleximeter;* the one striking the blow is called a *plessor.* Various forms of these have been designed, but the one depicted in the accompanying illustration is practically the only one in general use. The fingers are commonly used as plessor and pleximeter because they are more convenient, being always available. The second finger of the left hand is applied to the part under examination, and the blow is struck with the second or with the first and second fingers of the right hand. As to the relative value of instrumental aid in the practice of percussion, authorities give diverse opinions. One set tells us that the nicest points can only be elicited by the use of the plexor and pleximeter, while the other states that they are, at best, of doubtful value. It is more than probable that each is of more value than the other according to the individual peculiarities of the practitioner and the method to which he was educated. It is therefore the duty of all to become equally proficient in the practice of percussion with the fingers alone and with the aid of instruments. The latter may not always be at hand, in which case one must rely upon the plexor and pleximeter given him by nature.

The pleximeter should be applied firmly and evenly to the chest in the intercostal spaces.

The finger with which the blow is struck should be flexed at nearly a right angle, and, with a motion strictly from the wrist, the blow should be delivered perpendicularly to the surface of the pleximeter. The striking finger should be raised immediately upon the delivery of the blow; otherwise, the percussion-note will not be clear. The greatest care should be exercised that all blows delivered are of uniform force.

FIG. 77.



Plessor.

The force of the blow is a very important matter. As a general rule, the best results will be obtained by moderately light percussion. As the resulting sounds are due to vibrations of the walls of the chest, heavy blows must produce sounds over a widely-diffused area, and thus obscure the tones from the parts immediately beneath the pleximeter. Of course, one must be governed largely by existing conditions. If the chest or portions of the same are covered by large masses of fat or muscle, a relatively hard blow is required. If, on the other hand, the patient is a child or a greatly emaciated subject, light percussion will be essential. The employment of strong percussion to discover deeply-situated foci of consolidation is liable to give inaccurate results, because of the diffusion of sound due to the extensive area of the chest-walls thus set in vibration.

(*c*) **The Interpretation of the Sounds Elicited.**—The character of the sound elicited by percussion will vary according to the situation at which percussion is made. We may have sounds which have been respectively designated as dull or flat, resonant, tympanitic, and amphoric. Each of these sounds is normal, occurring in certain places to be designated shortly. When observed in other portions of the chest, they constitute positive evidence of organic changes.

The practised diagnostician, hearing a percussion-note, is enabled at once to assign it its proper quality, just as a finished linguist is able to converse in a foreign language without first formulating his ideas in his mother-tongue. The beginner cannot do this; he must think in his own language, and he must translate as he speaks. So it is with the interpretation of percussion-sounds. The beginner must not expect to recognize the nature of the sounds off-hand. He must observe their several qualities, and from these he determines their general character. The qualities of the percussion-sounds are pitch, intensity, and duration. When the novice percusses, he should inquire of himself the pitch, the intensity and duration of each, and thus he is able to say that there exists dullness, resonance, or tympany. To obtain empirical conceptions of the general qualities of percussion-sounds by percussing over localities where such exist is all right in its way; but it is apt to tend to routinism, and does not acquaint one sufficiently with the many nice variations which are perfectly consistent with health. It is impossible, by the mere use of words, to convey or give an accurate conception of the various qualities of chest-sounds; hence some arbitrary standard is necessary; but it must not be misused.

i. **Pitch.**—The determination of the pitch of a percussion sound is absolutely essential, as it bears important relations to dullness, resonance and tympany. High pitch attends dullness or flatness, and low pitch, tympany. Still, this rule is not invariable, for high pitch may accompany tympany.

ii. **Duration of the Sounds.**—The term duration is self-explanatory. It is long with resonance or tympany, and short with dullness or flatness.

iii. **Intensity.**—The intensity of the sound varies with the amplitude of the vibrations, and hence is greater when the pitch is low, and in the presence of tympany and resonance. It is also modified by the force of the blow.

We next have to consider the **various qualities of chest percussion-sounds,** namely, resonance, dullness, tympany, and amphoric resonance.

i. **Resonance.**—When the chest over healthy lung-tissue is percussed there is elicited a sound clear in character, of relatively low pitch, and of moderate duration and intensity. It exists in various degrees of purity in different portions of the chest. In any normal individual, it exists in its most characteristic quality in the second interspace in front, in the upper portion of the axillary region on the side, and below the spines of the scapulæ behind. Its pitch is somewhat higher over the right apex than over the left.

It is modified in health by several conditions. In persons with thick chest-walls, or over thick deposits of fat, over the scapulæ or mammary glands, it is impaired, and lacks clearness. Conversely, in persons with thin chest-walls, pulmonary resonance is unmistakable.

The variations in the elasticity of the chest-walls by reason of age or individual peculiarities also modify it. In the aged, whose chest-walls are rigid, it is lacking in clearness ; in children, it is full and clear.

It will be deduced from the above that there can be no arbitrary standard for normal pulmonary resonance. Each individual has his own standard. This must be determined by the examiner by means of careful comparison of symmetrical portions of the chest.

ii. **Dullness or Flatness.**—A typically flat percussion-note can be obtained by percussion of the thigh. It is of high pitch, low intensity, and short duration. A sound of similar quality may be elicited in the chest by percussion over that portion of the heart uncovered by lung substance on the left side ; and over the lower portion of the thorax on the right side where the liver comes in contact with the thoracic walls. Occurring in any other locality of the chest, it is significant of disease, for it indicates the absence of air from the tissues beneath. Dullness may be absolute or relative. Absolute dullness is readily determined by the merest tyro. Relative dullness requires considerable skill and experience for its discovery. It is found in those portions of the chest in which normal lung-tissue intervenes between a deeply-situated solid organ and the thoracic walls. Good examples of relative dullness are found in the areas surrounding the superficial cardiac dullness, and over an area for two inches above the upper line of absolute hepatic dullness.

iii. **Tympany.**—A tympanitic sound is characterized by its low pitch, long duration, great intensity, and a peculiar metallic quality which is readily recognized. It is found over large air-containing cavities with smooth walls. In the normal chest, it is found only over Traube's semilunar space. (See page 193.)

iv. **Amphoric Resonance.**—This is a low-pitched sound, resembling in its general features tympanitic resonance, already described. It may be obtained in health by percussion over the trachea ; also by striking the cheek distended with air.

v. **Cracked-Pot Sound.**—This sound is obtainable in health only when percussing the chest of a crying child. It is generally described as of clinking quality. It may be imitated artificially by closing one's hands together, palm to palm, with an air-space intervening, and striking the back of one hand against the knee. Air is thus forced out and produces a "clinking" sound.

chus, the lumen of which is free; in bronchiectasia; in pulmonary cavities which communicate with a bronchus and have thin elastic walls.

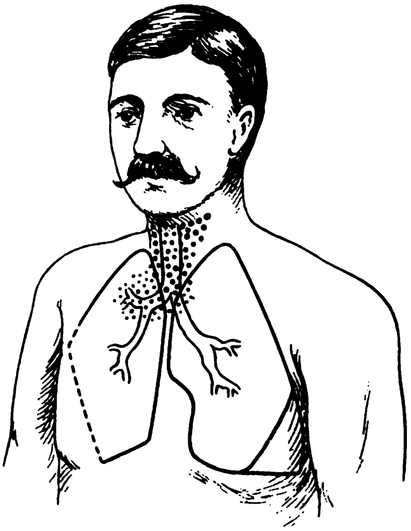
Amphoric Resonance.—Amphoric resonance is observed pathologically in pneumothorax, and over large superficial cavities with smooth walls, communicating with a bronchus.

Cracked-Pot Resonance.—This is elicited usually when there is a cavity near the apex communicating with a bronchus. It is important for its production that the chest walls be thin and yielding. The patient should be instructed to keep his mouth open. It will be heard more readily if the observer have the patient hold the bell of a stethoscope in front of his mouth, while he himself percusses and listens.

4. **Auscultation.**—Auscultation of the respiratory apparatus may be practised with or without instrumental aid. The former is called mediate, and the latter immediate auscultation. Each method has its advocates, the majority, however, favoring the use of the unaided ear. So far as the stethoscopes in use until within a comparatively recent period are concerned, there can be no doubt that the sounds of respiration are best studied with the observer's ear applied directly to the patient's chest. But when one becomes accustomed to the phonendoscope or Bowle's binaural stethoscope, he finds that he can hear more distinctly with their aid than without them. The main objection urged against instrumental help is that it may not always be at hand, in which case the physician must rely upon his ear, which, unfortunately, has not been trained for auscultation in this manner. We do not think much of this objection, as it is the physician's duty to be prepared for all emergencies when in attendance upon his patients. Another objection has been found in the many irrelevant sounds on first listening through a binaural stethoscope for the first time. It has been facetiously expressed that we hear too much. The majority of these extraneous sounds will not be present if the bowl of the instrument is applied evenly and firmly to the chest-walls. With experience, furthermore, other sounds will soon be disregarded. The facility with which it is possible to disregard sounds other than those upon which attention is fixed is found in one's utter obliviousness to heart-sounds when listening to the respiration, and *vice versa*. Again, these objections sink into insignificance when we consider the advantages to be derived from the use of the phonendoscope and its modifications. To lady patients, mediate auscultation is much more agreeable. The observer, likewise, finds it more pleasant, especially when examining patients whose cleanliness is not above suspicion. Physicians who tend to obesity find that with the stethoscope they can work in a comfortable attitude, and this of itself tends to accuracy of observation. Finally, there are areas of the chest,—as in the upper axillary and the supraclavicular region,—to which the ear cannot be directly applied.

The foreign sounds which are especially liable to prove a source of annoyance to the auscultator are the so-called skin-rubs, the crepitation of a hairy chest, and those produced by careless manipulation of the stethoscope itself. A little experience soon eliminates the first of these, as the practitioner learns to hold the instrument firmly and steadily in contact with the chest-walls. Without much care, he soon recognizes these superficial friction-sounds, and ignores them. The crepitation on the hairy chest may be obviated by moistening the hairs with water or oil. The mistaking of sounds originating in the contractions of chest-muscles for râles is to be obviated by experimental observation of the same, that one may become well acquainted with their peculiarities.

FIG. 78.



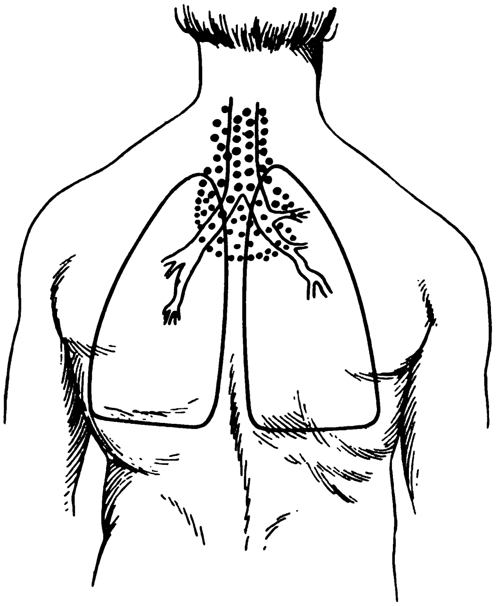
Illustrates normal location of bronchial (large dots) and broncho-vesicular (small dots) breathing in front of chest.

In the beginning of his practice, it is important that the auscultator take plenty of time for observation. The experienced can readily recognize alterations in the sounds in a few seconds. The novice must not expect to do this. On the contrary, he should listen patiently until he is sure that he has observed correctly. Continued attention to sounds makes them much more evident. Qualities which were at first indistinct become plain. Extraneous sounds which were at first annoying almost disappear.

As in the case of percussion, preparation of the patient is an all-important matter for the practice of auscultation. The patient's chest must be bared; at the most, it should be covered by a single layer of a thin, flexible fabric. When listening to the front of the chest, the patient should sit symmetrically erect, with his muscles thoroughly relaxed. When auscultating the back of the chest, his position should be that already advised as suitable for percussion of that area.

In many instances trouble is experienced by failure on the part of the patient to breathe correctly. He either attempts to show off by making deep, unnatural breaths, or he stops breathing altogether. These difficulties are soon righted, however, for in a few seconds the patient proceeds to breathe according to his natural method. Should this not be sufficient to bring out abnormal sounds, we may proceed to teach the patient to breathe deeply and forcibly.

FIG. 79.



Illustrates normal location of bronchial (large dots) and broncho-vesicular (small dots) breathing in back of chest.

The Normal Respiratory Sounds.—The respiratory sounds heard in the normal chest include

a. Bronchial or tubular breathing;
b. Vesicular respiration; and
c. Broncho-vesicular breathing, which is a combination of the first two named (a) and (b).

Each of these varieties of respiration is heard normally in specific portions of the healthy chest. Audible in other localities, they constitute positive evidence of disease. In the clinical study of these various types of respiration, it is important that attention be paid to the relative duration and intensity of the inspiratory murmur, their pitch and their quality.

(*a*) **Bronchial or Tubular Breathing.** — An adequate conception of bronchial or tubular breathing may be obtained by listening with the stethoscope over the trachea. The sound thus obtained is characteristically of tubular quality. It is distinctly separable into two portions, an inspiratory and an expiratory portion, of nearly equal lengths; if there is any difference, the latter is slightly the longer. Between the inspiratory and the expiratory acts is a distinct interval of silence. The pitch of the expiratory sound is somewhat higher than that of the inspiratory. The latter is slightly weaker in intensity than the former. Anteriorly it may be heard over the lower portion of the neck and the upper portion of the manubrium sterni; posteriorly, over the lower two cervical vertebræ.

(*b*) **Vesicular Breathing.**—To obtain a correct idea of vesicular breathing, the student should listen over a portion of the chest some distance from the trachea or primary bronchi, *e.g.*, in the axilla, or in the infraclavicular region. He will hear a sound of peculiar breezy quality, which has been compared to the rustling of the leaves moved by a gentle wind passing through a distant grove of trees. This sound is divisible into inspiratory and expiratory portions, not separated by an interval of silence as in bronchial respiration. The inspiratory portion is longer than the expiratory by the relation of three to one. It is low in pitch. The expiratory portion of the murmur presents an even lower pitch than the inspiratory, and is of more feeble intensity. In addition to the peculiar breeziness of inspiration, it presents a slight blowing quality.

Vesicular breathing is heard most distinctly, in addition to the regions above specified, below the scapulæ behind. It is audible in all situations excepting those mentioned as occupied by bronchial and broncho-vesicular breathing, but in some portions of the chest it is much weaker than in others.

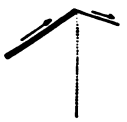
(*c*) **Broncho-Vesicular Breathing.**—This may be best described as a sound partaking of the qualities of bronchial and vesicular breathing combined. It is heard normally over the lower portion of the manubrium sterni and the costal cartilages at their sternal junctions at this level, and posteriorly over the upper three or four dorsal vertebræ and for a short distance to each side of the same. It is characterized by a certain harshness of the inspiratory act, while expiration becomes prolonged, and is louder than in health.

In children, the respiratory murmur is greatly increased over that observed in adults. Hence the term *puerile* has been applied to it.

Modifications of the Respiratory Murmurs in Disease.—The modifications of the respiratory sounds in disease include the following:

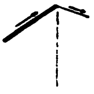
a. Alterations in intensity of the respiratory (vesicular) murmur.
  i. Weakness or suppression.
  ii. Exaggeration or puerile breathing.
b. Alterations of rhythm.

FIG. 80.



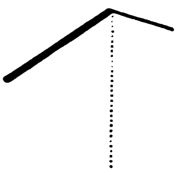
Vesicular breathing.

FIG. 81.



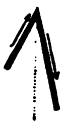
Distant vesicular breathing.

FIG. 82.



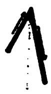
Exaggerated vesicular breathing.

FIG. 83.



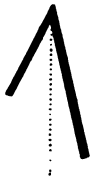
Bronchial breathing of moderate intensity.

FIG. 84.



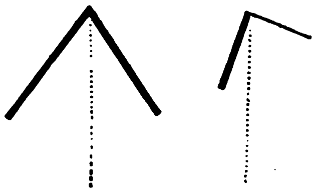
Distant bronchial breathing.

FIG. 85.



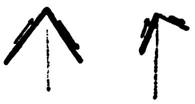
Very loud bronchial breathing.

FIG. 86.



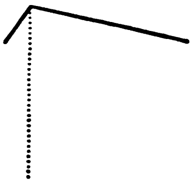
Two common types of bronchial breathing.

FIG. 87.



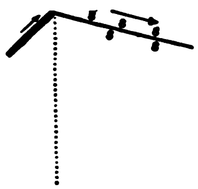
Distant broncho-vesicular breathing.

FIG. 88.



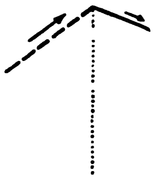
Emphysematous breathing.

FIG. 89.



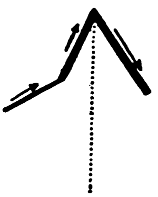
Asthmatic breathing.
s,s,s, squeaking râles.

FIG. 90.



Cogwheel breathing.

FIG. 91.



Metamorphosing breathing.

Note to figures 80 *to* 91, *inclusive.*—Cabot has systematized the graphic representation of respiratory types in the above symbols. The up-stroke represents the inspiratory murmur, and the down-stroke the expiratory. The relative duration of each of these acts is indicated by the lengths of the lines; their intensity, by the thickness of the lines. The pitch of the sound is represented by the angle which each of these up- or down-strokes makes with the perpendicular. With this explanation, the various features of the different types of respiration become clear on inspection of the above series of diagrams taken from Cabot's excellent work on "Physical Diagnosis."

i. Expiration prolonged.
ii. Interrupted or cogwheel respiration.
iii. Metamorphosing breathing.

c. The appearance of bronchial and broncho-vesicular breathing in portions of the chest in which they are normally absent.

d. Amphoric or cavernous breathing.

(*a*) **Alterations in Intensity of the Respiratory Murmur.**—i. *Weakness or Suppression.*—The conditions which may give rise to weakness or suppression of the respiratory murmur include: (1) Defective movements of the thorax; (2) the presence of fluid or air in the pleural cavity; (3) obstruction of the air-passages; (4) pulmonary emphysema; (5) pain in the thoracic walls.

In the majority of cases in which the respiratory murmur is weak, owing to defective movements of the thorax, the underlying cause is general ill-health or bad breathing habits. In some of these,—a goodly proportion, indeed,—tubercular changes have already taken place, but are discovered only with the greatest care. In another class of cases, there is actual loss of power of the respiratory muscles from central nerve lesions. These cases are rare, however.

The presence of fluid or air in the pleural cavities is responsible for the entire disappearance of the vesicular murmur over that portion of the chest occupied by the abnormal accumulation. It does this in two ways: First, by so compressing the lung on the affected side that no air can enter the same; and, secondly, by reason of the poor conduction of sounds through gaseous and liquid accumulations.

When a bronchus is obstructed by a plug of mucus, as in bronchitis, or by outside pressure, as in intrathoracic tumor, it is a natural sequence that the vesicular murmur over its tributary area be impaired or even completely abolished, according to the extent of the resulting obstruction. Absence of the respiratory murmur from this cause not infrequently escapes discovery because it is frequently associated with loud râles, which are apt to distract attention from the regular breathing sounds.

In pulmonary emphysema, the chest being in a condition of constant expansion, the excursion of the respiratory movements is more or less impaired, and consequently the respiratory murmur is diminished.

The enfeeblement of the vesicular murmur in painful affections of the thorax is dependent entirely upon the resulting interference with chest movements.

ii. *Exaggerated or puerile breathing* occurs pathologically over healthy lung in an attempt to compensate for loss of function of other portions of the pulmonary structures, as in pneumonia and pleurisy with effusion. In puerile breathing there is no regular rule as to the relative intensity of inspiration and expiration; sometimes one and sometimes the other is the louder.

(*b*) **Alterations of Rhythm.**—i. *Expiration Prolonged.*—Prolongation of the expiratory murmur is observed in two distinct classes of cases: one in which it constitutes the first evidence of a change of respiration type to broncho-vesicular breathing, and is therefore to be regarded as a sign of commencing pulmonary consolidation; and the other in which it is due to lack of elasticity in the pulmonary structures, as in emphysema, or to inability to expire, as in asthma. In the class first mentioned the change is a local one, and should be regarded as strongly suggestive of a commencing tuberculosis, of which it is commonly the initial physical sign. In emphysema and asthma, the change is evident throughout the entire chest. Inspiration is short and expiration is prolonged.

ii. *Interrupted or Cogwheel Respiration.*—In "cogwheel" respiration, the inspiratory portion of the murmur is interrupted by breaks, which transform the sound into a series of short puffs or jerks. Its clinical significance varies according to the extent of the phenomenon. When manifested throughout the entire chest, it is to be regarded as evidence of some general or nervous cause, usually fatigue, nervousness or chill. When local, it is strongly suggestive of a commencing tuberculosis. Its presence in the latter condition is explained by the interruptions to the entrance of air into the pulmonary vesicles by the occlusion of the bronchioles by tubercular deposits. Air is thus permitted to enter the different lobules at different times, and so causes the interruptions in the respiratory sound. Cogwheel respiration announcing the onset of tuberculosis is to be distinguished from the same symptom resulting from other causes by the associated prolongation of the expiratory murmur. With due care, one may also discover in the majority of the tubercular cases small, moist râles, especially after coughing or taking a full breath.

iii. *Metamorphosing Breathing.*—By this term is meant a breathing that is suddenly transformed from the vesicular to the bronchial or broncho-vesicular type, or in which there is a sudden accession to the intensity of the sound. Such changes are to be explained by the sudden displacement of a plug of mucus in a tube by the act of inspiration, so that the sounds through it become audible.

(*c*) **The Appearance of Bronchial and Broncho-Vesicular Breathing in Portions of the Chest in which they are Normally Absent.**—i. *Bronchial Breathing.*—Bronchial breathing, when heard in any other location than those indicated on page 384, is positive evidence of pulmonary consolidation. It is especially prominent as a symptom of croupous pneumonia and pulmonary tuberculosis. It may also be produced by hæmorrhagic infarcts and pulmonary syphilis.

Important as is bronchial breathing as auscultatory evidence of phthisis and pneumonia, its absence must not be assumed to exclude the possibility of these affections, for should a bronchus or a series of bronchioles be occluded by an inflammatory exudate, it will fail of conduction to the consolidated pulmonary tissue. It may also fail of recognition in certain rare instances in which the consolidation is centrally situated, the bronchial breathing being obscured by the vesicular sound of the surrounding lung-structure.

Bronchial breathing may occur in a respectable proportion of the cases of pleuritic effusion. The exudate in these cases is just sufficiently great to compress the vesicular structure of the lungs without obliterating the bronchial tubes. Hence bronchial breathing is produced. The sounds are ofttimes very feeble,—sufficiently so, indeed, to make their recognition doubtful by any but those who exercise great care in auscultation. It is usually heard most distinctly at the upper layer of the effusion, and close to the spine posteriorly.

ii. *Broncho-Vesicular Breathing.*—When audible in other localities than those indicated on page 384, broncho-vesicular breathing indicates a moderate degree of pulmonary consolidation, such as occurs in the early stage of pulmonary tuberculosis and the earliest and last stages of croupous pneumonia. It may also be audible over the retracted lung in cases of pleuritic effusion.

(*d*) **Amphoric or Cavernous Breathing.**—Amphoric breathing is always pathological. It is observed in cases of pulmonary tuberculosis with formation of cavities, and in pneumothorax. The respiratory murmur presents a hollow, empty sound, which has been not inaptly compared to the sound produced by blowing across the mouth of an empty bottle.

Pathological Sounds.—With the exception of amphoric breathing, the respiratory changes already described as occurring in diseased conditions are but modifications of normal types. In addition to these, however, are other sounds which are never present in health. These abnormal sounds may be included under the following heads:

a. Râles.
    i. Dry râles.
        i. Sonorous.
        ii. Sibilant.
    ii. Moist râles.
        i. Crepitant.
        ii. Sub-crepitant.
        iii. Large moist or mucous râles.
b. Friction-murmurs.
c. Metallic tinkling.

(*a*) **Rales.**—i. *Dry Râles.*—Dry râles have been divided into the *sonorous* and *sibilant.* Sonorous râles are characterized by their loudness and their low-pitched musical character. They are produced in the large bronchial tubes. Sibilant râles, on the other hand, are high pitched, and may be best described as possessed of a whistling quality. Râles of the above character may be observed in some cases of bronchitis, before the disease has advanced to the stage of exudation, being due to the swelling or tumefaction of the bronchial mucous membrane ; in asthma, as a result of the spasm of the tubes.

ii. *Moist Râles.—i. Crepitant Râles*—The crepitant râle is a fine sound, which has been compared to the crackling produced by the throwing of salt on a hot stove ; or to the peculiar sound produced by rolling a lock of dry hair between the fingers held close to the ear. So far as the sound itself is concerned, it is a dry râle ; but it owes its origin, so it has been claimed, to an exudate in the air-vesicles, the sound itself being due to the separation of the alveolar walls. Crepitant râles are heard during inspiration, especially towards the termination of the act ; they are not influenced by coughing. At one time crepitant râles were said to be pathognomonic of croupous pneumonia ; but we now know that they are sometimes absent in that disease, and may be present in other conditions. Crepitant râles may be observed in the following conditions :

1. The first stage of croupous pneumonia.
2. Pulmonary œdema.
3. Hæmorrhagic infarction.
4. Localized atelectasis.
5. Sometimes in the pulmonary apices of those who habitually under-inflate their lungs.
6. Hypostatic congestion of the lungs.

The crepitant râles which are heard along the thin margins of the lungs at their bases, when breathing changes from a superficial to a deep character, usually disappear after taking a few breaths.

Cabot is authority for the statement that crepitant and subcrepitant râles are quite common over the inferior margin of the normal lung at the bottom of the axilla. This statement he supports by the following figures : " Out of 356 normal chests, to which I have listened especially for these râles, I found 228, or 61 per cent., which showed them on one or both sides. They are very rarely to be heard in persons under twenty years of age. After forty-five, on the other hand, it is unusual to find them absent. In my experience, they are considerably more frequent ' over the sixth and seventh ribs in the axillary line ' than in any other portion of the lung, but they may be heard in the back, or elsewhere. In view of these facts, it seems to me that we must recognize that it is almost if not quite physiological to find the finer varieties of crackling râles at the base of the axillæ in persons over forty years old. I have supposed these râles to be due to a partial atelectasis, resulting from disease in the thin lower margin of the lungs.

Such portions of the lung are ordinarily not expanded unless the respirations are forced and deep."*

ii. Sub-Crepitant Râles.—These are also known as fine moist râles, a synonym which answers well as a definition. They originate in the smaller bronchioles, in the interior of which there is considerable secretion, which is set in vibration by the respiratory current. They are audible during both inspiration and expiration. When the conditions favoring their production are present, and yet are not audible, they may be rendered so by coughing or forced inspiration.

Sub-crepitant râles are heard in the following conditions:

1. Acute bronchitis in the stage of secretion.
2. Pulmonary œdema.
3. Pulmonary hæmorrhage.
4. Early stage of croupous pneumonia.
5. Stage of resolution in croupous pneumonia (râle redux).
6. In the first and second stages of pulmonary tuberculosis.

The value of sub-crepitant râles, localized at either apex as a sign of beginning tuberculosis, cannot be overestimated. They are then due to the localized bronchitis set up by the tubercular process.

Occurring at the bases of the lungs without other physical signs, and when few in number, they are suggestive of slight œdema, hypostatic congestion, or disuse of the lungs in part.

iii. Large Moist or Mucous Râles.—These are sometimes known as bubbling or gurgling râles; and this explains their character. They originate in the larger tubes, and are dependent upon the vibrations set up by the action of the respiratory air-current upon the mucous and other secretions therein contained. They are heard during inspiration or expiration, or both. They are observed in the following conditions:

1. Bronchitis.
2. The third stage of phthisis.
3. Pulmonary œdema.
4. Pulmonary cavities, when the passage of air through the same is interrupted by passing through fluid accumulations in the same.

Very coarse moist râles may originate in the trachea. They are usually quite loud, and may be heard at a distance from the patient. They are especially liable to occur in the moribund state, and hence are sometimes referred to as the death-rattle. They are especially common in the unconscious and those greatly prostrated by disease.

(*b*) **Friction-Murmurs.**—These are produced by the rubbing of opposing pleural surfaces against each other; it is essential to their production, however, that the pleural surfaces be roughened by a fibrinous exudate, as in pleurisy. It has been suggested that a conception of the pleural friction-sound may be obtained by holding the palm of one hand over one's ear and rubbing the dorsum with the fingers of the other. Clinically, friction sounds are characterized by their superficiality; by the fact that they are intensified by the pressure of the stethoscope; by their fixity in one locality, not being altered by coughing or forced respiration; by being audible during inspiration, but continuing into expiration. Friction-sounds sometimes disappear. They may then be reproduced by manœuvres suggested by Abrams: The arm on the corresponding side is raised over the head as in the performance of Sylvester's method for performing artificial respiration. Then auscultation is practised over the suspected area. "By this manœuvre the movement of the parietal against the visceral pleura is opposite in direction to that occurring during the respiratory act, and, for this reason, the pleuritic sound may often be elicited after it has been exhausted in the ordinary acts of breathing." The other movement is known as the "*decubital manœuvre.*" "Let the patient lie upon the affected side for a minute or two, then let him rise quickly and suspend respiration. Now listen over the affected area, at the same time directing the patient to take a full breath."

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* Cabot: "Physical Diagnosis of the Diseases of the Chest," p. 105 *et seq.*

With but few exceptions, pleural friction-sounds indicate the first stage of pleurisy. They disappear with the appearance of effusion. They may also occur over the pulmonary apices in phthisis. They may also be produced over the base of the right side of the thorax in hepatic or subdiaphragmatic abscess. Vierordt* is authority for their occurrence in "pyæmic deposits in the lungs, infarction, bronchiectasis with reactive pneumonia, and pleuritis with emphysema."

(*c*) **Metallic Tinkling.**—Metallic tinkling gives to the ear of the auscultator the impression of a dropping into a liquid confined in a cavity. And this, indeed, is its origin, for it is observed in hydro- and pyopneumothorax. It has also been suggested that metallic tinkling occurs in large pulmonary cavities.

Auscultation of the Voice-Sounds in Health and Disease.—When, in health, the ear is applied to the chest while words are spoken by the patient, audible vibrations will be transmitted to the chest-wall. We call this phenomenon the *vocal resonance.* It depends, for its production, upon the same physical conditions as those calling forth the vocal or tactile fremitus. It also possesses the same clinical significance as that phenomenon, except in rare instances, the rationale of which has not been explained.

In healthy chests, it is more prominent over the right than the left apex. It is more distinct in persons with thin chest-walls, and who have a bass voice; it is less prominent in women and children.

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* **Vierordt**: "A Clinical Text-Book of Medical Diagnosis for Physicians and Students." Fourth American edition, p. 201.

When examining the vocal resonance, the patient may use either his whispered or his ordinary conversational voice. He should utter the words "one, two, three" in a monotone. The information obtained by the whispered voice is much the more valuable; indeed, it has been claimed that increased resonance with the whispered voice is a more delicate sign of consolidation than is breathing.

In the case of the conversational voice, the sound heard by the ear is buzzing or purring; with the whispered voice, it is a feeble, blowing sound, resembling the ordinary expiratory murmur. It is heard distinctly in normal chests, with few exceptions, only over the trachea and primary bronchi in front and behind. In other portions of the chest it is weak, and may even be inaudible.

As a result of disease, vocal resonance may be increased or decreased. *Increased vocal resonance* is heard in all conditions in which there is pulmonary consolidation, excepting when there is associated obstruction of the bronchial tube tributary to the affected area. Such increase in vocal resonance is known as *bronchophony*, because of its similarity to the voice-sounds as heard over the main bronchi and trachea. When the sounds become so distinct that it seems as if they emanated from the portion of the chest immediately beneath the stethoscope, the phenomenon is known as *pectoriloquy*. If the patient speaks slowly, the articulation is clearly discerned. Pectoriloquy is observed over cavities communicating with a large bronchus; over consolidations in communication with large bronchi; in the case of pneumothorax having free communication with a bronchus; and, though rarely, over compressed lungs above a large pleural effusion.

It is now universally conceded that the use of the whispered voice is capable of giving much more information than that of ordinary conversation. This is explained by the fact that a whisper practically amounts to a forced expiration. Modifications of it may be produced by areas of consolidation much smaller in size than those capable of producing tubular breathing.

Diminished vocal resonance occurs whenever a bronchial tube is obstructed, whether from accumulation of secretions within or pressure of a tumor from without. It is also observed in pleural effusion and when the pleura is greatly thickened, and in some cases of dense pleuritic adhesions.

Egophony is a modification of the vocal resonance characterized by a tremulous bleating sound, which has been compared to the crying of a goat. It is observed, though rarely, in cases of moderate pleuritic effusion. The best location for eliciting this sign is at or below the angle of the scapula. Taylor asserts that egophony may also be observed in some cases of pneumonia, when the bronchial tubes are obstructed by plugs of fibrin.

Brief Descriptions of the Symptoms and Clinical Course of the Various Respiratory Diseases.

The various diseases of the respiratory system may best be studied clinically by classifying them as follows:

1. **Acute Diseases.**
    (a) With dullness on percussion.
    (b) Without dullness on percussion.
2. **Chronic Diseases.**
    (a) With dullness on percussion.
    (b) Without dullness on percussion.

Acute Diseases.

(*a*) With Dullness on Percussion.

The acute pulmonary disorders attended by dullness on percussion include the following:

1. Croupous pneumonia.
2. Catarrhal pneumonia.
3. Pleurisy with effusion.
4. Pulmonary congestion.
5. Pulmonary œdema.
6. Abscess of the lung.
7. Pulmonary gangrene.

1. Croupous Pneumonia; Pneumonic Fever; Lobar Pneumonia.

Although pneumonic fever presents, in the majority of cases, a typical course, in which the various symptoms are associated with each other in a characteristic manner, and the sequence of clinical events is remarkable for its regularity, nevertheless there is no acute specific disease capable of producing as great a variety of clinical pictures, unless we except typhoid fever.

The typical case starts with severe rigor or chill, which, however, is usually of short duration. The temperature rises at once to a high point, and continuing, with slight remissions, until the crisis, which takes place usually between the seventh and eleventh days, but exceptionally as early as the third. Coincident with the fever are numerous other symptoms, some due to the profound constitutional disturbance, and others indicating the localization of disease in the respiratory apparatus. Among the former are rapid pulse, severe headache, general pains, and vomiting; and of the latter, cough, at first dry, later with expectoration, severe pain in the side, rapid respiration, and active movements of the alæ nasi. We now proceed to a study of the individual symptoms.

The pulse is rapid from the first, usually disproportionately so for the fever. In persons of robust constitution at the time of the attack, its strength usually remains good throughout the illness. With individuals already weakened by chronic disease, alcohol, or old age, weakness and rapidity are characteristic from the incipiency of the attack.

The course of the fever has already been fully described in the chapter on "Temperature" (page 48), and therefore requires no further description here.

Pain in the chest is an early phenomenon, and is the cause of considerable suffering. It is due to an associated pleurisy. It is nearly always located in the chest. Very exceptionally, it may occupy anomalous locations, or may be referred to the abdomen, and so give rise to diagnostic errors if the physical signs are not discovered.

Respiratory disturbance is an almost necessary part of the symptomatology of pneumonia. Great rapidity of breathing—from 30 to 60 per minute—is observed. Respiration is usually impeded by the thoracic pain to which reference has already been made. In some cases there may be actual dyspnœa.

The *cough* in the early stage is dry. With the advent of the second stage it becomes frequent, and is attended by expectoration of a thick, viscid sputum, which is ofttimes blood-streaked. Exceptionally the blood may be present in sufficiently large quantity to be called a hæmorrhage. More frequently it is mixed thoroughly with the mucus, constituting the oft-described "rusty sputum" of pneumonia. After the crisis the cough becomes much easier and expectoration is no longer difficult, the sputum thereafter being for the most part of muco-purulent character. Certain atypical cases of pneumonia are remarkable for the almost entire absence of cough and expectoration. Such latent pneumonias are especially apt to occur in the aged, and in the victims of alcohol and Bright's disease. In low types of the disease the sputum, instead of being tenacious, is thin and dark-colored, and has not inaptly been described as "the prune-juice expectoration."

While the condition of the heart has no bearing on the diagnosis of pneumonia, attention to it is of the highest importance as indicative of the patient's progress and general condition. The interference with the pulmonary circulation throws increased labor upon the right heart. The second sound at the pulmonary area becomes accentuated. It is this symptom which should be most carefully watched; for, should the second pulmonic sound weaken without corresponding evidence of improvement in the condition of the lungs, we may well fear that the right heart is giving out and dilatation of its cavities will follow. While, on the other hand, the maintenance of the accentuated pulmonic second sound indicates strain upon the right heart, it also shows that that organ is for the present, at least, able to withstand the extra pressure.

In no pulmonary disease are the physical signs more characteristic than they are in pneumonic fever. *Inspection* demonstrates nothing of importance beyond the rapid or embarrassed respiration. There may be a slight increase in the circumference of the affected side, but it is discoverable only by careful measurement. *Palpation* discovers, besides a lack of local expansion, increased vocal fremitus over the consolidated lung. When, however, the pneumonia is what is known as massive, and the bronchial tubes are filled up with secretions, the vocal fremitus will be found to be diminished.

Percussion discloses in the first stage of the disease a slight dullness over the consolidated lobe, and this becomes more manifest as the lesions progress, until finally it may even amount to absolute flatness. The dull quality of the percussion-note is sometimes masked in the stage of engorgement by reason of the possession of a tympanitic quality. This same semblance of tympany may be resumed when the lesions are undergoing resolution. Exceptionally, percussion-dullness may be noteworthy because of its absence. This is ofttimes the case in the pneumonias of children, and in adults when the consolidation is centrally located. When the lesion involves the lower lobe of the left lung, the dullness may be masked by reason of a tympany transmitted from the adjacent distended stomach or colon. When dullness is present, its extent must not give rise to false ideas as to the extent of the lesion ; for example, the upper limit of the lower lobe posteriorly is about on a line with the middle of the scapula, but the dullness resulting from its consolidation may cover the entire back on the corresponding side.

Auscultation early demonstrates the presence of the crepitant râles. This may not be readily manifested, because of the usually associated quiet, suppressed breathing. Their absence cannot be assured unless the patient draws a long breath. The vesicular murmur then becomes harsh. When percussion-dullness is manifested, the breathing becomes tubular. During the stage of resolution, râles of any degree of coarseness and fineness appear (*râle redux*). Vocal resonance is increased.

The symptoms above detailed are those upon which we rely for the diagnosis of pneumonia. Characteristic as are the physical signs, they must not be relied upon exclusively, for circumstances may cause unusual modifications ; or, as has already been shown, they are sometimes absent in part. Clinical accuracy therefore demands that they must always be studied in conjunction with the rational symptoms.

But pneumonia may be attended by numerous symptoms which are not essential to the diagnosis of the disease. Knowledge concerning them is of importance because of the possibility of their confusing the diagnosis and directing attention to organs other than the lungs. Especially does this remark apply to the cerebral symptoms. A fairly large number of cases of pneumonia are associated with delirium, which sometimes amounts to actual mania. Indeed, such cases have been diagnosed as acute mania, the pulmonary consolidation being discovered only at the autopsy. It may also happen that an associated meningitis is an early phenomenon, and if the chest is not examined critically,—a very difficult thing to do under the circumstances,—the primary pneumonia may escape recognition.

Pneumonia occurring in alcohol habitues is liable to run an atypical course. Cough, expectoration and fever are not prominent features; hence attention is not directed to the chest. At the same time, pneumonia in such subjects is very prone to be the means of exciting delirium tremens. It is a good rule, therefore, to systematically examine the chests of such patients as frequently and as thoroughly as circumstances will permit.

The digestive symptoms include a moist but very heavily-coated tongue, vomiting in the early stage, and enlargement of the liver and spleen.

Aside from the usual characteristics observed in fevers, the urine shows great diminution in chlorides. This symptom seems to run parallel with the progress of the case, and hence may be utilized to a certain extent as a prognostic sign.

The principal blood change noted in pneumonia is a leucocytosis. The count usually runs from 14,000 to 40,000 per cubic millimeter; exceptionally, it may reach 100,000. The absence of leucocytosis may be regarded as an unfavorable sign.

Pneumonia is interesting because of the numerous complications. These include pleurisy, pericarditis, simple and malignant endocarditis, meningitis, multiple neuritis, gastric disturbances, jaundice, hemiplegia, and renal disease.

In addition to certain atypical forms of pneumonia to which incidental reference has been made in the preceding pages, attention may be directed to still others, especially the terminal and secondary pneumonias. Under the terminal pneumonias may be included those cases which occur in the subjects of renal disease, pulmonary tuberculosis, heart diseases, etc. Inasmuch as the symptoms are by no means obtrusive, such cases are apt to fail of recognition, excepting on the post-mortem table.

The secondary pneumonias are especially apt to follow or complicate the infectious diseases, and, like other atypical varieties, present but few of the characteristic symptoms of pneumonic fever. Even the physical signs are obscure.

2. Catarrhal Pneumonia; Broncho-Pneumonia.

Cases of catarrhal pneumonia may be divided into two classes—primary and secondary. The former occur in children almost exclusively; the latter as a sequence or continuance of a variety of conditions, among which are acute bronchitis, the infectious fevers, and the entrance of foreign particles into the respiratory passages (*aspiration* or *deglutition pneumonia*). Of the infectious fevers thus acting as a causative factor, the chief ones are measles, whooping-cough, diphtheria, and scarlatina. In these diseases it is a most important cause of death. The deglutition pneumonias occur in persons who have been rendered unconscious from such diseases as uræmia or cerebral hæmorrhage, thus permitting of the entrance of portions of liquid or solid food into the larynx, and finally the smaller bronchi. Akin to the latter cases are the pneumonias succeeding pulmonary hæmorrhage, bronchiectasia, and ruptured empyema.

Most cases, however, result from exposure to cold, with or without a preceding bronchitis. The early symptoms are therefore those of acute bronchitis, but with more marked constitutional disturbances, as fever and rapid pulse. We also note the phenomena indicating involvement of the respiratory apparatus, as rapid respiration, shortness of breath, cough with expectoration, and the physical signs.

The fever presents no definite type. The temperature range is very irregular, but rarely, excepting in children, does it run over 103° F. The variations from day to day, or even from hour to hour, are irregular.

The pulse is rapid, especially in young subjects.

The respiration-rate may run from 50 to 80. The shortness of breath sometimes amounts to actual dyspnœa, with evidence of deficient aëration, as cyanosis.

The cough, which, during the preceding bronchitis, had been loose and painless, with the extension of the disease to the pulmonary lobules, becomes dry and ineffectual. Later there is some expectoration, which is usually muco-purulent, but may be blood-streaked.

Inasmuch as the pulmonary changes consist of scattered foci of consolidation, percussion should reveal patches of localized dullness. As a matter of fact, the consolidated areas are usually so small as to make their recognition, clinically, entirely out of the question in the vast majority of cases. Hence it is that percussion avails but little in the diagnosis of catarrhal pneumonia, unless, indeed, we regard the absence of marked dullness an important feature when taken in conjunction with the severity of the associated symptoms. On auscultation, numerous patches of fine râles; exceptionally, when the consolidated areas are closely grouped, we may hear tubular breathing; but in the majority of cases the only disturbance of the vesicular murmur is the assumption of a harsh character.

In fatal cases respiration becomes more and more labored, and the heart shows signs of failing. The disease has no definite duration; acute cases continue all the way from ten days to three weeks.

There is a class of cases of catarrhal pneumonia in adults in which the constitutional disturbance is rarely great. The prevailing symptoms include irregular fever, with cough. Physical examination discovers patches of consolidation and small râles. Such cases, though offering a favorable result so far as the immediate future is concerned, are prone to end in pulmonary tuberculosis.

3. Pleurisy with Effusion.

The typical case of acute pleurisy is ushered in by fever with or without a chill, and more or less severe pain in the side. There may be some cough; but if so, it is of a dry, harassing character. There is some embarrassment of respiration, but it is due entirely to the pain occasioned by the respiratory movements. As the disease progresses, effusion takes place. The inflamed pleural surfaces are no longer in contact, and the pain in the side ameliorates. The dyspnœa increases, however, now being due to the impairment of the pulmonary space by the fluid accumulation. The severity of the dyspnœa seems to be due more to the rapidity of fluid accumulation than to the actual quantity. To relieve the pain, which is aggravated by every movement of the affected side of the chest, the patient lies on the affected side. The usual duration of a case of pleurisy is from one to two months.

Atypical cases come on slowly, the usual clinical phenomena, as fever and pain in the chest, not being at all obtrusive. It is not unusual for the patient to keep at his ordinary work, knowing that he is not in his usual good health, but not being able to specify anything especially wrong.

In any case, the physical signs establish the diagnosis. In the first stage, the friction-murmur, especially marked towards the close of inspiration, becoming more marked with increase of stethoscopic pressure, tells the nature of the case with unerring precision. With the appearance of effusion the friction-murmur disappears. Then there is dullness on percussion, sometimes amounting to absolute flatness over the fluid accumulation. The effusion brings about certain changes, evident to inspection. Thus, the affected side of the chest appears to be unduly rounded or bulging, although by mensuration the enlargement is not as great as it appears. The depressions between the ribs are lessened. Displacement of adjacent organs is very common, varying, however, according to the side of the chest involved. The heart in left-sided and the liver in right-sided pleurisy are the organs principally affected in this respect. Some popular notions concerning the upper limit of the percussion-dullness require correction. It is not on a horizontal line, as generally supposed, but describes a modified letter "S." Nor is the area of dullness as readily modified by position as ordinarily stated. Decubitus does produce some alteration, but the change takes place with comparative slowness. With the absorption of the effusion the percussion-dullness disappears. It is not unusual for some impairment of the pulmonary resonance to remain, by reason of a permanent thickening of the pleura.

With the beginning of effusion, auscultation shows a progressive enfeeblement of the respiratory murmur, and this may eventuate in absolute silence over the effusion. Sometimes there may be heard, if care is observed, feeble and distant tubular breathing. Vocal and tactile fremitus are impaired over the effusion. Above the line of effusion, and on the healthy side of the chest, the vesicular murmur is exaggerated (*compensatory breathing*).

Some cases of pleurisy proceed no farther than the first stage (*dry pleurisy*).

Patients who have had pleurisy are exceedingly liable to develop tuberculosis in after years.

Some cases do not undergo resolution, and as a result we have chronic fibrinous pleurisy or empyema (*purulent pleurisy*).

So far as life is concerned, the prognosis of pleurisy is, as a rule, favorable. Occasionally death results from a complication, as endocarditis, pericarditis, meningitis, embolism, and thrombosis, all of which are serious.

4. Pulmonary Congestion.

Pulmonary congestion may be either active or passive. **Active pulmonary congestion** seems to be a theoretical pathological conception rather than a distinct clinical entity. Active hyperæmia is, of course, the initial change in many pulmonary diseases, but it is very doubtful if its clinical recognition is possible. As a temporary condition, it may be produced by the inhalation of irritating fumes, very hot or very cold air, and inflammation of adjacent structures. The symptoms which have been assigned to it include dyspnœa, cough with stitching pains, blood-stained expectoration, feeble respiratory murmur, and some impairment of percussion-resonance.

Passive congestion, also known as **hypostatic congestion** and **hypostatic pneumonia,** is, on the other hand, frequently observed clinically, due to well-defined causes, producing a typical symptom-group. The lesion is located usually at the bases of the lungs, and is due to the combined effects of the long-maintained recumbent posture and impaired heart action. It is especially liable to occur in the aged, the victims of long-continued fevers, mitral disease, dilatation of the right heart, and cerebral hæmorrhage. In many cases it produces no prominent symptoms, and is to be recognized only on physical examination. Thus, there is dullness on percussion over the affected region, with feeble respiratory murmur and moist râles. When the congestion is well advanced, there will be cough, dyspnœa, and even hæmoptysis. The prognosis is that of the disease or condition upon which it is engrafted.

5. Œdema of the Lungs.

This is, in the majority of cases, a terminal event in the course of a number of diseases having a fatal tendency. The symptoms of cases of this character consist of dyspnœa and cough. The latter has a peculiar ineffectual sound, and, when successful in bringing up sputum, the latter is clear and frothy. There is impaired pulmonary resonance, and auscultation discloses the presence of loud, moist râles in the larger and medium-sized tubes. Pulmonary œdema is usually of acute onset, and ends fatally within a few days. Cases occurring in the course of chronic Bright's disease are apt to appear suddenly.

6. Abscess of the Lungs.

Pulmonary abscess itself is an acute lesion; but it is a secondary process which may be engrafted upon acute or chronic conditions, which may be enumerated as follows: (1) Croupous pneumonia; (2) Pulmonary tuberculosis; (3) Bronchiectasia; (4) Pyæmic infarctions; (5) Pulmonary embolism; (6) Perforation of the lung by malignant disease of the œsophagus; (7) Suppuration of bronchial glands; (8) Empyema rupturing into the lung; (9) Perforation of the lung by mediastinal abscess; (10) Perforation of the lung by a sub-diaphragmatic abscess; (11) Perforating wounds of the lung; (12) Suppuration from a hydatid cyst. The symptoms are those of the primary affection plus the constitutional disturbances attendant upon suppuration, expectoration of a large quantity of pus, and certain physical signs. The constitutional symptoms include fever of septic type; the temperature is usually high. It may or may not be associated with rigors. The initial expectoration of pus is unexpected, and is very free. Microscopic examination discovers that it contains lung-tissue. Previous to this event, a diagnosis is largely conjectural. There will be, it is true, a localized dullness on percussion, with absence of vesicular murmur; but such a condition is liable to suggest a diagnosis of pleural effusion, from which it may be differentiated by the absence of visceral displacements and the preservation of the vocal fremitus. The many positions which it is possible that a pulmonary abscess may occupy, and the many directions in which it may extend and finally perforate, suggest the widespread symptomatology possible, and the difficulties attendant upon the diagnosis. The expectoration in the beginning is not offensive. Later, especially if drainage of the cavity be inefficient, the fœtor may be very pronounced.

7. Pulmonary Gangrene.

This is always a secondary process, succeeding one of the following conditions: Putrid bronchitis, bronchiectasia, aspiration pneumonia, especially in the paralyzed and insane, acute and chronic pneumonia, pulmonary tuberculosis, septic embolism, extension of disease from adjacent viscera, diabetes, chronic alcoholism, and Bright's disease. The essential symptom is a horribly offensive expectoration, the sputum separating into three layers, as follows: "a greenish-brown heavy sediment; an intervening thick liquid, which sometimes has a greenish or brownish tint; and on top a thick, frothy layer."* It always contains portions of pulmonary tissue. The physical signs are not likely to be distinctive, owing to the varying conditions surrounding the lesion. In a general way, they may be stated to be those of pulmonary cavity plus those of the primary disease. Without the fœtid sputum a diagnosis is impossible, and this may happen by reason of the plugging of a bronchus leading to the affected area. Pulmonary gangrene is inevitably fatal.

(*b*) Without Dullness on Percussion.

The acute pulmonary diseases which do not have dullness on percussion as a symptom include—

1. Acute catarrhal bronchitis.
2. Acute miliary tuberculosis.
3. Acute dry pleurisy.

1. Acute Catarrhal Bronchitis.

Acute catarrhal bronchitis is, in the majority of cases, the result of "taking cold," especially in the predisposed. As a rule it is preceded by catarrhal inflammation of the upper respiratory tract. It may also occur as part of the clinical features of certain infectious diseases, notably influenza, typhoid fever, and measles.

The cardinal symptoms of acute bronchitis are cough, expectoration, and dyspnœa. Cough, however, is the prominent symptom. In the initial stage it is dry and hacking. With the advent of the stage of secretion it becomes loose, and is accompanied by the expectoration of sputum, at first clearly mucus, and later muco-purulent. In the majority of cases the dyspnœa is mild; often not sufficiently marked to attract attention. In gouty subjects and the victims of alcoholism and nephritis it is a prominent feature. Subjective discomfort referred to the chest consists of a sense of oppression referred to beneath the sternum, and indicates involvement of the medium-sized tubes. Constitutional disturbance is usually mild, consisting, for the most part, of slight fever (temperature 100° to 102° F.), some slight acceleration of the pulse, and weakness.

Physical signs are not infrequently absent in the mild cases. In others, percussion gives no change in sound, excepting in those instances in which there is a localized collapse of air-cells from obstruction of the bronchioles. Auscultation may give entirely negative evidence, or the vesicular murmur may be somewhat harsh. Sometimes, by reason of bronchial obstruction by the secretions, it may be entirely absent until the tubes are cleared by coughing. Râles are not uncommon. In the first stage they are dry and squeaking, and not influenced by coughing. In the fully-developed disease they are of the large and small moist varieties. In children and the aged,—in whom bronchitis is always to be regarded as a serious disease,—they are usually loud, and are audible over the entire chest.

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* Osler: "Practice of Medicine." Fourth edition, p. 661.

2. Acute Miliary Tuberculosis.

Careful study of cases of acute miliary tuberculosis of the lungs will show, in the majority of cases, a tuberculous focus in some portion of the body, the breaking down of which is responsible for the acute process. Hence, we find in this disease a history of a former pulmonary lesion, now latent, or tuberculosis of glands or bones. The initial symptoms are those of a diffuse bronchitis; the local and constitutional symptoms are disproportionately severe to the physical signs. Cough is a prominent symptom. It may be dry and hacking, or it may become moist with expectoration of a muco-purulent character. Sometimes it is attended by hæmoptysis. In rare cases it may present the rusty appearance observed in pneumonic fever. Dyspnœa is always a prominent feature; in fact, it has been said that no pulmonary affection, other than pneumonia and emphysema has this symptom more fully developed. The patient is usually cyanosed, the discoloration being particularly manifested in the lips and tips of the fingers. Fever is always present, the temperature usually ranging around 103° F., and not infrequently presenting the inverse type. The pulse is weak and disproportionately rapid as compared with the temperature. As the case progresses to a fatal termination the complexus of symptoms which we know as "the typhoid state" becomes manifest.

The *physical signs* relate almost exclusively to the evidence afforded by auscultation. Owing to the small size of the tubercular granulations, as a rule the percussion-note is unaltered. The vesicular murmur is harsh and expiration is prolonged. Râles include the sub-crepitant, crepitant, sibilant and sonorous varieties. In the later stages of the disease the râles become coarser.

The diagnosis of acute miliary tuberculosis of the lungs should not be attended by any difficulty. Its suddenness and its rarity always make it an unexpected condition; hence it is not usually recognized as promptly as we would expect. From *typhoid fever*, it is to be differentiated by the rapid pulse, the embarrassed respiration, and the cyanosis. *Acute bronchitis* never presents the profound constitutional and local disturbance observed in miliary tuberculosis.

3. Acute Dry Pleurisy.

Dry pleurisy presents the same symptoms as the first stage of pleurisy with effusion.

Chronic Diseases.

(*a*) With Dullness on Percussion.

The chronic pulmonary diseases presenting dullness on percussion include the following:

1. Pulmonary tuberculosis.
2. Tumors of the lung.
3. Empyema.
4. Hydrothorax.
5. Hæmothorax.
6. Interstitial pneumonia (pulmonary fibrosis).
7. Brown induration of the lungs (chronic congestion).
8. Atelectasis.

1. Pulmonary Tuberculosis.

Pulmonary tuberculosis may be divided into three classes as follows:

i. Caseous tuberculosis.
ii. Fibro-caseous tuberculosis.
iii. Fibroid tuberculosis.

i. Caseous Tuberculosis; also known as Acute Pneumonic Phthisis, Phthisis Florida, Galloping Consumption.

Clinically and pathologically two varieties of caseous tuberculosis may be recognized, namely, the disseminated or broncho-pneumonic and the lobar varieties. Caseous tuberculosis represents but a small percentage of the total cases of pulmonary tuberculosis, the lobar form being especially rare. It may occur in persons who already have chronic tuberculosis of the lungs, or it may appear as a primary affection.

The onset of the disease may be either gradual or sudden. In the latter case, there are fever with rigors, rapid pulse and respiration, cough, and possibly bloody expectoration. This combination of symptoms presents a strong resemblance to pneumonia, with which disease it is usually confounded in the beginning. Before long, however, the fever assumes the well-known hectic type, and is associated with night-sweats, rigors, and general emaciation. The sputum is at first mucoid, and possibly blood-stained, but soon becomes muco-purulent, and in turn purulent. Microscopic examination nearly always discloses the presence of the tubercle bacilli.

The physical signs resemble those of broncho-pneumonia. Loss of pulmonary resonance is observed, especially at the apices. This is associated with crepitation. As the disease progresses, complete dullness may be noted. Auscultation also discovers diffuse râles of bubbling, crackling, or metallic quality. The respiratory sounds assume a tubular quality. Bronchophony is often present. In the lobar variety the symptoms simulate those of croupous pneumonia from the beginning, and no suspicion of the mistaken diagnosis will be held until the time for the crisis has arrived and that fortunate event fails to materialize.

It would seem that cases of acute pneumonic phthisis can only be recognized with certainty in the beginning by microscopic examination of the sputum, which should contain not only the tubercle bacilli but portions of broken-down lung-tissue.

II. Fibro-Caseous Tuberculosis; Chronic Ulcerative Tuberculosis; Chronic Pulmonary Tuberculosis.

This is the most common variety of pulmonary tuberculosis. The sequence and character of lesions in this disease give a clew to its clinical features. The rational symptoms are necessarily those of disturbed pulmonary function. The starting-point is almost invariably in the upper portion of the apices of the lungs, and nearer to the posterior than to the anterior surface. In the beginning it is a deposit of miliary tubercles, followed by pulmonary consolidation,—really a tubercular broncho-pneumonia. Then there ensues caseation, with or without ulceration and formation of a cavity. In some cases there is a formation of connective tissue. These various changes are generally a matter of many months from their incipiency to their termination in death. Ofttimes the progress of the disease is checked in various portions of its course, to remain quiescent for prolonged periods.

The onset of fibro-caseous pulmonary tuberculosis is always gradual. In fact, it is at times difficult to state exactly just where the illness had its commencement. In some of the cases the onset may be so insidious that the pathological changes are already well advanced before the patient considers it worth while to consult a physician. In other cases the initial symptoms are not referred to the lungs, the patient complaining of general weakness and prostration, or possibly some disturbance of digestion. The only symptom other than those obtainable by physical examination of the chest which would suggest pulmonary tuberculosis is afternoon or evening rises of temperature, possibly with subnormal morning temperature. In another class of cases the patient is in fair health, and does not regard himself as sickly, when he is seized with pulmonary hæmorrhage, and from that time on is never well. Cases of this character have led to the theory of *phthisis ab hæmoptoë*. Lastly, we have the cases which constitute the majority; that is, those in which the pulmonary tuberculosis seems to be the direct continuance of an attack of acute bronchitis, or of repeated attacks of that disease,—generally, too, in persons who have been more or less careless, or who take cold readily.

The cardinal symptoms of chronic pulmonary tuberculosis include cough, expectoration, tubercle bacilli in the sputum, thoracic pains, fever, emaciation, anæmia, and the physical signs attendant, according to the stage of the lesion, upon a localized bronchitis, pulmonary consolidation, and pulmonary destruction (cavity formation). These symptoms necessarily vary in prominence and in their associations according to the mode of onset and the manner of progress of the case. To them may be added other clinical phenomena by reason of direct and indirect effects of the tubercular process upon other viscera.

Cough.—This is an early and constant symptom,—the one which forces itself upon the attention of the patient and drives him to the physician for relief. In the beginning, it is of a dry, hacking character. At first it is only annoying at night, or after undue exertion ; later, it becomes constant. This dry cough soon changes to one of moist character, attended by expectoration, which will vary in individuality according to the stage of the disease ; starting as purely mucoid, it progressively becomes muco-purulent and purulent.

The *sputum* will change in quantity and appearance according to the mode of onset of the disease and the stage to which it has advanced. When the disease has supervened upon chronic bronchitis, it is profuse from the first. Even during the stages in which it preserves its catarrhal characteristics, careful search will discover more or less numerous small puriform masses ; and it is these which should be searched most diligently for the presence of the tubercle bacilli. With the appearance of tissue destruction the sputum becomes purulent, and its color of a greenish or greenish-yellow color. Sometimes it forms flattened purulent masses (nummular sputum), and it is then said to be a sign of the presence of cavities. The daily quantity of sputum may vary all the way from what may be described as scanty to six or eight ounces in the cases in which the disease is well advanced and is progressing rapidly. Not infrequently it is more or less mixed with blood, which sometimes appears as clots and sometimes as mere streaks. Microscopic examination of the sputum, the technique of which will be described elsewhere (Chapter XII.), will discover the presence of the tubercle bacilli, and, if destructive changes are taking place, elastic fibres from the lung.

Pain may be experienced in any portion of the chest, but it is far more likely to be referred to the parts overlying the apices than elsewhere. In the early stages it may be described as a tenderness on pressure. Occurring in the lower portion of the chest, it is to be regarded as evidence of localized inflammation of the pleura. Lesions secondary to the pulmonary tubercular process may also excite pain.

The *fever* of pulmonary tuberculosis has been described in a preceding chapter (p. 61). It is only necessary to remind the reader, at this time, that it is a fair indicator of the activity of the lesions.

Emaciation is almost always present, some slight loss of weight being noted from the first. So important is this symptom, that its absence is suggestive of the disease in hand being other than tuberculosis. Nevertheless, one meets occasionally with cases in which the bodily weight is maintained at a normal standard for some time. Emaciation is brought about by a combination of causes, including deficient aëration, the drain of a destructive lesion, the fever, and the digestive disturbances.

Respiratory disturbance is not as prominent as the extent of the pulmonary lesions would permit one to expect. This is explained by the slow progress of the disease. Usually we find some increase in the respiration-rate, and shortness of breath after slight exertion. In the latest stage, when extensive destruction of lung-tissue has taken place, there may be actual dyspnœa.

The *pulse-rate* is quickened because of the general weakness and the fever. In the terminal stages it is very rapid, weak, and of low tension.

The *digestive symptoms* include indigestion in the initial stages, vomiting and diarrhœa. The indigestion is of especial importance from a therapeutic standpoint, because maintenance of nutrition is a necessary element in the successful treatment of pulmonary tuberculosis, and we cannot secure this until the stomach has been placed in good condition.

Diarrhœa is usually a late symptom, although it is not infrequent in the beginning as a result of dietetic errors.

The physical signs of pulmonary tuberculosis vary according to the stage of the disease during which the patient is examined. In the fully-developed disorder they are so distinct, and present in such numbers, that they may be recognized by the tyro. In the incipient disease they are ofttimes so delicate, and require such painstaking care for their discovery, that only the experienced can properly interpret them. It seems to the writer that the prominence of the signs in the developed disease gives the uninitiated a false conception of the clinical picture. If he does not find obtrusive phenomena, he is inclined to belittle the importance of the apparently insignificant abnormalities present, and thus he will fail of a diagnosis. In not a few cases he is obliged to be satisfied with persistent slight fever not to be otherwise accounted for, associated with loss of flesh and debility. As to the physical signs of the earliest stages, they include the presence of small, moist râles at the pulmonary apices, diminished elasticity of the chest, or *localized* and *persistent* cogwheel respiration. At this early stage no changes will be evident by palpation or percussion. So far as signs and symptoms are concerned, the case is one of a mild bronchitis localized at the apices.

The next stage of the disease is characterized by consolidation, the bronchial condition still persisting. Hence we have added to the signs already noted those usually attendant upon consolidation, namely, more or less marked dullness on percussion, increased vocal resonance, broncho-vesicular and tubular breathing.

The many lesions of the advanced stage of pulmonary tuberculosis cause the physical signs to be equally varied, including, as they do, the evidence of catarrhs, consolidation, cavities, etc. It is, however, the evidence of advanced consolidation and disintegration of lung-tissue that is characteristic of this stage. These are so prominent, as has been before stated, that the beginner in physical diagnosis can recognize them. What was mere impairment of resonance or dullness in the previous stages becomes almost complete flatness ; the tactile fremitus, as evidence of the consolidation, becomes especially evident ; and this remark applies also to the vocal fremitus. The breath-sounds are equally characteristic of consolidation, as we find tubular breathing predominating ; in many localities there will be well-marked broncho-vesicular breathing. Aside from the catarrhal condition of the tubes, we have an additional source of secretions in the destruction of tissue. Coarse râles are therefore audible. In other portions of the lungs there will be crepitant and sub-crepitant râles, especially the latter.

As to cavity formation and its recognition, the problem is not as easy a one as usually stated, if entire dependence is placed upon the physical signs. When the signs which are stated to be the evidence of cavities are present, then the diagnosis is undoubted ; but, unfortunately, the physical conditions are by no means always present ; hence, cavity formation is not to be diagnosed off-hand. To be recognizable, a cavity must be sufficiently large, and it must have rigid walls and be in communication with a bronchus. If it happens to be filled with secretions, the physical signs will be those of consolidation. Its presence, then, is to be recognized only when empty. The signs upon which we are accustomed to rely for the discovery of cavities include amphoric breathing, cracked-pot resonance, and coarse, gurgling râles.

iii. Fibroid Tuberculosis.

This form of pulmonary tuberculosis is remarkable for its slow course, which is commonly interrupted by periods of arrest. It is apt to attack subjects of more mature years than the other varieties, and men more than women. Those who follow occupations exposing them to a dusty atmosphere are predisposed. The onset of fibroid phthisis is always insidious. For many years the patient may complain of nothing more than chronic cough with some loss of weight, and recurrent attacks of bronchitis, attended at times by asthma. Naturally the lungs become emphysematous, and this, in turn, increases the tendency to fresh bronchial catarrhs and asthma. Hæmoptysis is not uncommon, even in the early stages. The entire course of the disease is remarkable for the absence of fever. The pathological changes may be well advanced without producing much constitutional disturbance or marked emaciation. The ill-health seems to have a closer relation to the bronchitis and the emphysema than to the tuberculosis.

The prominent physical signs include retraction of the supra- and infra-clavicular regions, with impaired expansion, impaired resonance, weakness of the respiratory sounds or broncho-vesicular breathing, and fine crackling râles which may be widely distributed. If cavities form, they are usually "dry." Tubercle bacilli may be found in the sputum, but only after repeated examinations.

2. Tumors of the Lung.

The majority of tumors of the lung consist of either carcinomata or sarcomata, and are secondary to similar growths in other portions of the body. They are especially common in later middle life. A tumor confined to the lung-tissue itself gives rise to no symptoms other than those of disturbed pulmonary function and the constitutional manifestations incident to the growth, including dyspnœa, cough, expectoration, emaciation and fever. It is thus seen that for a considerable time, at least, the case is apt to be mistaken for bronchitis or consolidation. A suspicion that the diagnosis may be incorrect may be suggested by the disproportionately severe dyspnœa. As soon, however, as the tumor encroaches upon the mediastinum, the diagnosis of the case is not difficult. The recognition of these cases is at times rendered more difficult by the growth setting up secondary processes, as inflammation, in which the symptoms of pneumonia will be manifested. The diagnosis is rendered certain by the discovery of a prominence on the chest-wall, the constitutional cachexia, the history of the case, and the physical signs of consolidation without the presence of the tubercle bacilli in the sputum. The average duration of life in cases of pulmonary tumor is but a little more than thirteen months.

3. Empyema.

The symptoms of empyema depend in great measure upon the origin of the case. When, as frequently happens, the purulent accumulation is secondary to a sero-fibrinous pleurisy, the initial symptoms are those of that affection, the change to a purulent exudate being ofttimes associated with the constitutional symptoms of suppuration, including rigors, fever, leucocytosis, weakness and pallor. In other cases, the infection follows one of the acute infectious diseases, especially scarlatina, typhoid fever, pneumonia, measles and whooping-cough. Once in a while cases in which local infection serves as the cause are observed, as perforating wounds of the pleura, and malignant and tuberculous disease. Very exceptionally, cases seem to originate as a primary purulent pleurisy, especially in children.

Purulent pleurisy may begin either suddenly or insidiously. In the former case we have the constitutional disturbance of suppuration. The latter class of cases is the larger, and no symptoms other than the physical signs, some slight cough, dyspnœa, and leucocytosis, may be observed. Empyema is sometimes associated with pulsation (*pulsating empyema*).

Empyema requires mechanical treatment for its relief. In some few cases relief may be secured by absorption, especially in children, or by perforation of the lung or of the chest-wall.

4. Hydrothorax.

Hydrothorax is never a primary affection, the effusion developing in the course of such diseases as Bright's disease, cardiac incompetency (as part of a general dropsy), and in intrathoracic tumors which interfere with the pleural circulation. Most of the attendant symptoms are those of the primary affection, associated with those of pneumonary compression. The nature of the case can only be determined by physical examination, which will give the signs of pleural effusion. The affection is bilateral. At first respiration is but slightly disturbed; later, there are dyspnœa and cyanosis.

5. Hæmothorax.

Etiological factors play an important part in the diagnosis of hæmothorax, as hæmorrhage can only take place into the pleura as the result of erosion or injury of a blood-vessel. Thus, it may occur from the rupture of an aneurysm, cancer, tuberculous ulceration, and traumatism; or from constitutional diseases, as scurvy. The fluid accumulation taking place rapidly, there is marked disturbance of pulmonary function, as intense dyspnœa. There are also the general symptoms of hæmorrhage, as syncope, pallor, etc., and the physical signs of pleural effusion. The latter are not apt to be recognized, because the patient's condition is such as to make the necessary interference unwise.

Hæmorrhagic pleurisy covers a different class of cases, in which there is a pleural effusion which is admixed with blood. It occurs in conjunction with cancer, Bright's disease, and tuberculosis.

6. Interstitial Pneumonia; Pulmonary Fibrosis.

The term "interstitial pneumonia" is restricted to those cases of increased formation of pulmonary connective tissue occurring independently of tuberculosis. Such cases may arise as a direct sequel of pneumonia, rarely of pleurisy; to inhalation of dust, and to syphilis. The fibrotic changes are described as of two varieties: one in which the changes are massive and invade one lung (*lobar form*), and the other in which the lesions are disseminated (*broncho-pneumonic form*). Pulmonary fibrosis runs a very chronic course, cases of fifteen to twenty years' duration not being uncommon. The entire symptomatology seems to be included under cough, with some slight dyspnœa. The expectoration is usually profuse and muco-purulent in character. Hæmoptysis occurs in more than one-half the cases. On inspection, the affected side of the chest is markedly retracted and shrunken. The extensive contraction of pulmonary tissue causes the heart to be displaced towards the affected side. When the left is the affected side, a larger portion of the heart comes in contact with the thoracic wall, and its area of impulse is therefore increased. Percussion shows dullness over the affected side, excepting in such places as where there may be a sacculated bronchus, as in the axilla, and hyper-resonance over the sound side. The audible phenomena include râles of all varieties, and sometimes friction-sounds; breathing is commonly weak, and broncho-vesicular or bronchial in quality, especially over the apices; over bronchiectases it is amphoric or cavernous; at the base the respiratory murmur is weak, and is associated with small, moist râles. The main difficulty in the diagnosis of this affection is its differentiation from fibroid tuberculosis. Interstitial pneumonia is commonly unilateral, the opposite lung being sound. Fibroid tuberculosis will almost invariably show some lesion, at least at the apex of the better lung. Cases of the latter disease, moreover,—and this, after all, is the crucial test,—will show the presence of tubercle bacilli in the sputum.

7. Brown Induration of the Lungs.

This is a condition in which the connective tissue of the lungs is thickened, and there is associated deposit of pigment-granules. It is due to the long-continued passive congestion of the lungs in cases of mitral stenosis. It may, however, occur in other cardiac diseases. The disease is not readily recognized, because the symptoms are disguised by those of the primary disease. The physical signs are identical with those of passive congestion.

8. Atelectasis.

Atelectasis, or collapse of the air-vesicles, may occur as the primary condition, as in infants at birth; or it may be secondary to obstruction of the air-tubes, compression of the lungs as from pleural effusion, and intrathoracic tumors. A frequently-observed variety of atelectasis is observed in the case of persons who are ill with typhoid fever and bronchitis, in which the collapse takes place in scattered foci, especially at the lower borders of the lungs.

The congenital variety seems to originate in causes which depreciate the constitution of the child, as syphilis, scrofulosis, premature delivery, etc., and injuries sustained during parturition. The child presents shallow breathing and a feeble cry. In the acquired condition the symptoms of the primary disease make the recognition of the atelectasis at times a difficult matter. The physical signs include absence or weakness of the respiratory murmur over the collapsed area, and, if the latter is sufficiently large, impairment of the percussion-resonance. It can readily be seen how these can be obscured by the signs of the causative disease.

(*b*) Without Dullness on Percussion.

The chronic diseases of the lungs not attended by dullness on percussion include the following:

1. Chronic bronchitis.
2. Bronchiectasia.
3. Emphysema.
4. Asthma.

1. Chronic Bronchitis.

A history of causes capable of producing chronic bronchitis is an important though not a necessary factor in the diagnosis of the affection. These causes include frequent attacks of acute catarrhal bronchitis, old age, Bright's disease, gout, and climatic influences. Bronchitis may also occur as secondary to other respiratory disorders, notably to emphysema, tuberculosis, and valvular (especially mitral) disease.

The symptoms include cough with expectoration, some interference with respiration, and the physical signs. Constitutional disturbance is not observed excepting in far-advanced cases and in those associated with other serious diseases, as nephritis, etc. In the mildest cases the cough is observed only in the winter months (*winter cough*). Each year it is worse than the preceding one. It is then usually unattended by expectoration or any severe disturbance of the general health. The repetition of attacks leads to emphysema and interference with cardiac function, these, in turn, acting to increase the predisposition of the bronchial inflammation.

In another class of cases the patient suffers from a morning cough, with muco-purulent expectoration. In those of still more severe type the cough becomes more or less constant, and, by reason of the serious changes set up in the heart and lungs, the interference with the general health is very evident. Indeed, there may finally ensue emaciation, prostration, night-sweats, and continuous dyspnœa.

The respiratory disturbances range all the way from a slight breathlessness on any exertion,—breathing being comfortable when the patient is at rest,—to severe and constant dyspnœa. Respiration may be so embarrassed that the patient is obliged to assume the upright position, and the cutaneous surface exhibits a cyanotic discoloration. Sometimes the dyspnœa assumes the shape of asthmatic paroxysms, which occur at night.

The expectoration may be scant, as in *bronchitis sicca*, or it may be so profuse as to merit the designation of *bronchorrhœa*. In character it may be mucoid, muco-purulent, or even purulent. It rarely contains blood.

The physical signs in uncomplicated cases refer to auscultation only. They include moist râles, with feeble respiratory murmur. The râles are heard most readily over the posterior aspect of the chest. They differ from those of incipient pulmonary tuberculosis in being diffuse,—not localized. Percussion-resonance is normal. When, however, there are associated emphysema or collapse of the air-vesicles, we have the signs appropriate to those conditions,—hyper-resonance in the former, and impairment of percussion-resonance, if not actual dullness, in the latter.

After a number of years chronic bronchitis is apt to eventuate in well-marked emphysema, dilatation of the right heart, and visceral congestions. The course of the disease is not infrequently interrupted by acute bronchial or pulmonary inflammations.

Plastic bronchitis is a rare affection, rendered chronic by the liability to recurrence, and characterized by the symptoms of bronchitis as above outlined, but differing from that affection in that, four or five days after the onset of the paroxysm, there is an expectoration of a cast of the "bronchial tree," and this is followed by prompt relief of all the symptoms. Hæmoptysis occurs in about one-half the cases. The prognosis as to recovery from individual attacks is good; but, despite all treatment, the paroxysms recur over a period of many years.

Fœtid bronchitis is characterized by offensive sputum. It may occur independently of bronchiectasia.

2. Bronchiectasia.

Dilatation of the bronchial tubes may be either saccular or cylindrical or fusiform. It is necessary for its production that some changes in the bronchial walls must take place. As determining causes, we find those agencies which bring increased pressure to bear upon the weakened structures, and we find these in bronchial stenosis, as from stricture, accumulation of secretion, and fibrous thickening of the interlobular and intervesicular connective tissue, and tumors of the lung. Dilatation may also originate in traction on the tube by connective tissue in the surrounding lung-structure, as from interstitial pneumonia and collapse of the lung.

The characteristic symptoms include cough at comparatively long intervals, attended by profuse expectoration. The mucous membrane of the affected parts is somewhat insensitive; hence secretions are permitted to accumulate until they overflow and irritate healthy mucous membrane, when they are expelled by coughing. The sputum is not infrequently possessed of an offensive odor, which has been compared to that of decayed cabbage. The coughing paroxysms usually take place morning and evening. The physical signs are scarcely such as to make the condition recognizable during life. The characteristic lesion—dilated bronchi—should produce the phenomena of phthisical cavity. This it rarely does, because the cavity is seldom sufficiently large to be recognized. Even then it is necessary that the examination be conducted at a time when the cavity is empty. The signs upon which we rely include cracked-pot resonance, amphoric breathing, gurgling râles, and bronchophony. Bronchiectasia may be mistaken for phthisis. The cavities, however, are found mostly at the base of the lung, while in phthisis they are situated at the apex. Dullness on percussion is an early phenomenon of phthisis ; it is late or absent in bronchial dilatation.

3. Emphysema.

The term " emphysema " has been applied to a number of clinical conditions, as follows : (1) Extravasations of air into the interstitial pulmonary tissues, and known as *interlobular emphysema.* (2) Dilatation of the air-vesicles, known as *vesicular emphysema.* This, in turn, may be subdivided into other varieties, including *compensatory*, *hypertrophic*, *atrophic*, and *acute vesicular emphysema.* When we speak simply of emphysema in our clinical work, we refer to the hypertrophic or *large-lunged* variety ; and it is this which we now proceed to describe.

The two important factors in the production of emphysema include a predisposition which produces a weakness of the vesicular walls, the origin of which is not understood ; and the determining causes, which include numerous agencies which throw extra pressure upon the pulmonary structures. Among the latter may be mentioned repeated attacks of acute bronchitis, chronic bronchitis, asthma, and occupations the principal part of which is " blowing," as in musicians who play on wind-instruments, glass-blowers, etc. The disease is one of gradual onset, and presents, as its characteristic symptoms, cough with expectoration, gradually progressive dyspnœa, and ultimately cyanosis, the patient throughout the course of the disease being subject to repeated attacks of bronchitis, which, in turn, aggravate the original condition.

The *dyspnœa* at first amounts to nothing more than shortness of breath on slight exertion. Later it becomes constant, and is associated with pronounced wheezing. It is always increased during the intercurrent attacks of bronchitis, and may lead to the supposition that the patient is suffering from an attack of asthma. In the final stages of the disease the patient is obliged to sit up in order to breathe (*orthopnœa*).

The *cyanosis* appears at a stage of the disease when the patient is still able to go about ; in fact, as Osler has so graphically depicted, in no disease is there such marked cyanosis with ability of the patient to go about his ordinary duties.

The diagnosis of emphysema, after all, depends upon the physical signs. Of these, the alteration in the shape of the chest is of the highest importance. The antero-posterior diameter of the chest becomes lengthened so that it equals the transverse diameter, the sternum and intercostal cartilages become unduly prominent, the shoulders stooped, and the curve of the spine rounded. Such a chest has been described as "barrel-shaped." Such a name may be appropriate in that it looks like a barrel as much as it does anything else; but, at the same time, we would observe that to compare it to a barrel requires an active imagination. Percussion-resonance is remarkably clear,—abnormally so. The overlapping of the heart by the dilated pulmonary structures causes a diminution in the area of superficial cardiac dullness. The respiratory murmur is greatly weakened; indeed, in some cases it may be much obscured by the associated bronchitic râles. Expiratory murmur is considerably prolonged, and is often wheezy and harsh.

The cardiac action is ultimately disturbed in emphysema. At first the only sign relating to this organ is accentuation of the pulmonary second sound. Later the right heart undergoes dilatation.

Emphysema is incurable. It runs a prolonged course. The patient usually succumbs to one of the intercurrent ailments to which this disease makes him liable.

Atrophic or small-lunged emphysema is a form of senile atrophy. Its presence during life is to be inferred rather than asserted in decrepit, thin-chested persons of advanced years, who have long been sufferers from cough with shortness of breath.

Compensatory emphysema is a conservative process consisting of dilatation of healthy lung-structure in cases in which portions of the pulmonary tissue are temporarily or permanently rendered functionless by disease.

4. Asthma.

A variety of conditions serving to produce paroxysmal dyspnœa, unfortunately for clinical accuracy, have generically been called asthma. Qualifying adjectives serve to indicate the cause and fix the nature of the case. Nevertheless, such nomenclature is confusing, though at times convenient. For accuracy's sake, only those cases characterized as "spasmodic" should receive the designation "asthma."

The basic causes of most cases is found in a neurotic temperament. Heredity is sometimes a factor. It is not so much that asthma attacks a number of members of the same family as that the disease occurs in patients giving a family history of neuralgia, hysteria, and other neuroses. Some cases start in early childhood and continue to adult life; others, more fortunate, escape its suffering after puberty. No disease exhibits greater peculiarities respecting the agencies which excite a paroxysm. Some patients find relief at the sea-shore, others in the mountains; some have it at the sea-shore, but not while on the ocean; some suffer in a fog; others are relieved thereby; and so we might go on describing innumerable peculiarities respecting the sufferers from asthma. Fright, emotions, irregularities of diet, exposure to cold, etc., have all been assigned as exciting causes. Many cases occur in the subjects of naso-pharyngeal disease, and make absolutely perfect recoveries when the latter condition is cured.

The cardinal symptom of spasmodic asthma is paroxysmal dyspnœa, the main difficulty in breathing being experienced during the expiratory act. The attack may be ushered in suddenly without prodromes, or it may be preceded by certain symptoms, the nature of which is well recognized by the patient. Usually, the dyspnœa comes on at night, rousing the patient from sleep. Starting with a slight want of breath, there is in a few minutes a high-grade of dyspnœa. The respiratory efforts made to get breath are painful to witness. All the muscles of respiration are called into play. Expiration in particular is labored, prolonged and wheezy. The influence of the defective aëration is soon manifested by profuse sweat, weak pulse, and cold extremities. After lasting a varying length of time, the attack disappears, the patient coughing up large quantities of thick, stringy mucus, which has been compared in appearance to boiled tapioca. Examination of the sputum discovers small, tough masses, which, when carefully examined, are found to be Curschmann's spirals, and contain the Charcot-Leyden crystals. (Chapter XII.) Succeeding the paroxysm the patient is exhausted, and may go to sleep; or a second seizure may follow closely in the wake of the first.

The frequency of asthmatic seizures is very variable. Some patients have them night after night for an extended period. Then they fail to recur for months. Others suffer only in response to certain stimuli, which are characteristic for the individual case. Others have them whenever they contract a "fresh cold."

The physical signs of asthma include loud wheezing respiration, the expiratory movement especially being prolonged. Notwithstanding the violence of the movements, the vesicular murmur is not readily heard, not only because of its weakness, but also on account of the many associated rhonchi and râles present. Percussion shows no impairment; on the contrary, there may be hyper-resonance.

Special varieties of asthma have been designated as "cardiac," "renal," or "uræmic." They differ from true asthma in that the difficult respiration is not expiratory.

CHAPTER XII.

TRACHEA, BRONCHI, AND LUNGS.

THE symptoms produced by lesions of the trachea, bronchi, and lungs, include cough, expectoration, interference with respiration, and certain objective symptoms known as the physical signs. Interference with respiration has already been studied in Chapter IV., and the physical signs of pulmonary disease in Chapter XI.

Cough.—Cough is a reflex act excited by irritation of the terminal fibres of any of the branches of the pneumogastric nerves, consisting of a closure of the glottis, followed by a forcible ejection of air by reason of contraction of the muscles of respiration. As a rule, it is to be regarded as a conservative symptom, being designed to clear the respiratory passages of the source of irritation. The irritation must be of the respiratory mucous membrane. This is well illustrated by the fact that lesions outside of the bronchial tubes and its ramifications, *e.g.*, tubercular nodules and calcareous deposits, do not provoke cough. A certain amount of irritability of the mucous membrane is necessary. In health, or conditions approximating to it, the presence of mucus or a foreign body is sufficient to produce a paroxysm of coughing, which continues until the offending substance is expelled. In typhoid conditions and diseases of the brain in which the sensibilities are obtunded, this irritability does not exist, and coughing is not produced. The production of cough also demands a certain degree of strength, for the act is impossible in certain conditions of asthenia. Such a cessation of cough always leads to the accumulation of secretions in the respiratory passages, and is to be regarded as decidedly unfavorable, especially in pulmonary tuberculosis and pneumonia.

The sensations which excite coughing consist for the most part of a tickling sensation, which may be located in any portion of the respiratory tract, though especially in the upper, and of a peculiar stuffiness or oppression difficult to describe, which is referred to the chest. These sensations are intensified by all agencies which disturb quiet respiration.

The many possible causes of cough entail upon the examiner the greatest care in the examination of patients suffering with this symptom. It may be that there is no disease present, but that the patient's surroundings expose him to an irritating atmosphere, that is, one containing dust or noxious gases. The occurrence of cough under such circumstances is not at all surprising. While, in the majority of cases, cough is symptomatic of a respiratory disease, cases are not unusual in which the digestive system is at fault, as in those instances in which the act follows the ingestion of unduly hot or pungent food. It may also result from a too hearty meal. The primary disease may be in the nose or pharynx, the discharges from which drop into the larynx, thus exciting a cough apparently primarily laryngeal in origin. Irritation of any of the terminal filaments of the pneumogastric nerves in the abdominal or thoracic viscera may likewise excite cough.

Cough may be studied clinically as dry and moist; as constant or paroxysmal; as organic or reflex.

A *dry cough* is one produced by irritation of the terminal pneumogastric fibres, but is unattended by expectoration. It does the patient no good, and is usually a source of great annoyance. It sometimes interferes with sleep, and adds to the patient's exhaustion.

A dry cough is observed in the early stages of all bronchial and pulmonary affections, *i.e.*, before the stage of exudation in pleurisy, in which disease it is associated with stitching pains in the chest; it is the characteristic cough arising from reflex irritation in various portions of the body, and in the course of hysteria.

Reflex coughs may originate in the ear, the stomach, and the teeth. The ear-cough is a dry, irritating cough, and is most commonly observed during the manipulation of the external auditory meatus incidental to examination of that structure. Cases are observed in practice in which foreign bodies or impacted cerumen have excited it.

The so-called *tooth-cough* is observed with especial frequency in infants during first dentition, and in older subjects who have carious teeth.

The *stomach-cough* sometimes occurs in association with mild cases of gastric catarrh. It may be produced also by the upward pressure of an overloaded stomach. There is very good reason to believe that this cough is due directly to the catarrhal state of the pharynx usually observed in conjunction with gastric disorders.

The *nervous coughs* occur for the most part in hysterical and neurotic subjects. To this class belong the barking cough of puberty, the short barking cough of hysteria, and the hacking habit-cough of many nervous individuals. The dry barking cough is sometimes a prominent symptom in neurotic subjects possessed of the idea that they are victims of hydrophobia.

A *moist cough* is characterized by the free expectoration usually of mucus or muco-pus, although it may consist of pus, blood, or other discharges from the respiratory mucous membrane. It is to be regarded as a conservative symptom, as it rids the air-passages of its pathological secretions. The irritation giving rise to it disappears on free expectoration. A moist cough occurs in fully-developed bronchitis, tuberculosis, pneumonia, bronchorrhœa, bronchiectasia, emphysema, and the terminal stage of asthmatic seizures.

Constancy of cough indicates constancy of cause. Such a cough is strictly of pulmonary origin, and occurs in conjunction with bronchitis, phthisis, pleurisy, and pulmonary consolidations.

A *paroxysmal cough* may be of either pulmonary or nervous origin. In any case the source of irritation must be recurrent. In the case of pulmonary lesions, such a cough suggests that a certain quantity of secretion must accumulate before cough can be excited. Thus, in bronchiectasia or pulmonary cavities there will be long intervals of freedom followed by cough, with expectoration of large quantities of sputum, after which relief ensues. Such coughs are especially liable to appear in the morning on waking. They may occur during the daytime.

Whooping-cough is characterized by paroxysms of cough, in which the act ends by a long-drawn inspiration called the "whoop." This disease bears a close symptomatic resemblance to acute bronchitis, from which, however, it cannot be positively differentiated until the whoop appears. It may be suspected by reason of its obstinacy to treatment and the knowledge of the fact that the patient has been exposed to the contagium. It is very often associated with retching and vomiting.

A *cough with retching and vomiting* may also occur in tuberculosis, bronchitis, or other pulmonary diseases with irritable pharynx. It is not uncommon as an early morning symptom in alcoholic habitues with gastric catarrh.

Cough which is brought on *by change of position* may be due to the increased respiratory effort required in the exertion, or it may suggest alteration in position of a pleural effusion.

A *night* or *evening cough* may suggest bronchial catarrh or early pulmonary tuberculosis.

A *suppressed cough* indicates that the act is painful, and occurs in painful affections of the thoracic walls and acute pleurisy.

A "*winter cough*" is indicative of mild cases of chronic bronchitis. Expectoration is usually scanty and mucoid in character. It occurs for the most part in gouty subjects past middle life. It recurs with remarkable persistence year after year, tending to get worse with each attack. Finally it remains permanent.

The *cough associated with heart diseases* may be due to the pressure of a distended pericardium upon a bronchus or upon the left pneumogastric nerve, or to congestion of the lungs from defective cardiac action.

The *cough of aneurysm* may be bronchial or brassy, or simply dry and hacking. When the tumor presses upon the recurrent laryngeal nerve, the cough is dry and hacking.

Disease of the mediastinum produces a cough of dry and brazen quality.

The *cough of œsophageal disease* arises from associated pressure upon a bronchus.

Notwithstanding the prominence and importance of cough as a symptom, it is sometimes absent in diseases in which it ought to be present. Conditions associated with impaired irritability of the pulmonary tissues explain this anomaly. For example, in pneumonia we find cough absent not infrequently when that disease occurs in the aged, or in sufferers from alcoholism and chronic Bright's disease.

Long-continued coughing over a period of time gives rise to certain secondary changes. Thus it may produce bronchiectasia, owing to the pressure exerted upon certain weakened portions of the bronchial walls; emphysema, as a result of the pressure upon the air-vesicles; and to hæmoptysis, when there are vascular degenerations, congestion, or ulceration. When hæmoptysis exists, the persistence of coughing serves to keep it up.

Accidents in Coughing.—Paroxysms of coughing may, either by reason of their violence or weakness of special tissues, produce annoying or serious accidents. Thus, in women suffering from certain forms of uterine disease, coughing has excited uterine hæmorrhage. Subconjunctival hæmorrhages may take place in the victims of whooping-cough. Retinal hæmorrhages have likewise been reported as occurring from this cause. In persons with degenerated arteries, intracranial hæmorrhage has been excited from violent coughing. Before the latter can take place, there is usually observed turgescence of the face. In the aged and insane, in whom the bones have become unduly brittle, fracture of bones, especially of the clavicles, have been produced by the muscular strain incidental to coughing.

Expectoration.—By expectoration or sputum is meant all those substances removed from the air-passages by the acts of coughing and hawking. These substances are not always brought up with equal degrees of readiness. As a general rule, the deeper the situation in the respiratory passages from which they are derived, the greater is the difficulty of expectoration.

Sputum may consist of serum, mucus, muco-pus, pus, or blood. These constituents are readily recognized by the naked-eye appearances. Microscopically, the sputum may be found to contain epithelium, portions of lung-structure, and numerous micro-organisms.

Serous or *albuminous sputum* presents a clear or frothy appearance, and is of thin consistence. It occurs mainly in œdema of the lungs. It is observed also after paracentesis for pleural effusion.

Mucous expectoration is clear and glairy or viscid. It is observed in the early stages of acute catarrhal bronchitis, and in asthma. In the latter affection, the viscidity is often very marked. It consists chiefly of mucus and a few epithelial cells.

Muco-purulent expectoration presents a thick consistence and a yellow color. It consists of a mixture of mucus and pus. Under the microscope it is found to consist almost entirely of pus-cells. It is the characteristic sputum of fully-developed acute bronchitis, catarrhal pneumonia, and of pulmonary tuberculosis before there is any disintegration of lung-structure.

Purulent sputum presents a green or yellowish-green color. Its presence is indicative of suppurative conditions. Hence it suggests empyema discharging through a bronchus, pulmonary abscess, bronchiectasia, and cavity formation in the course of tuberculosis.

Quantity of Expectoration.—The amount of sputum discharged varies within broad limits. Thus, there may be but a few drachms, as in mild bronchitis, while in empyema discharging through a bronchus, or after paracentesis thoracis, it may reach two or three pints in twenty-four hours. Such copious expectoration, however, rarely continues for any extended period of time. The quantity is large also in bronchiectasia. In this disease, however, it is usual for cough and expectoration to be in abeyance until the bronchial cavities are filled; hence the characteristic symptom is cough, with copious expectoration at long intervals.

Odor.—Ordinarily sputum presents no odor. It is always offensive, however, in pulmonary gangrene, and very often so in bronchiectasia, bronchorrhœa, and pulmonary tuberculosis with cavity formation.

Color.—*Transparency* of sputum indicates its *serous* or *mucous* nature; a *yellowish* color, that it is *muco-purulent*; and a *green* or *yellowish-green*, that it is purulent.

An *admixture of red*, especially occurring in streaks, suggests the presence of blood.

A *rust-color* (which is due to blood) is observed in the sputum of croupous pneumonia.

Prune-juice sputum is due to the presence of altered blood in the expectorated materials. It occurs late in the course of the septic forms of croupous pneumonia, and in gangrene and cancer of the lungs. It is to be regarded as an unfavorable sign.

Currant-jelly sputum occurs in cancer of the lung, and occasionally as a manifestation of hysteria.

A *dark-brown sputum*, which has been compared to *anchovy-sauce*, is observed in conjunction with the rupture of amœbic liver abscess into the lung. The suspicion is changed to a certainty by the discovery of the parasite under the microscope.

A *peculiar yellowish or greenish color* may be due to mixture with biliary coloring matters. This may occur in the course of jaundice; or, in the absence of that symptom, it is suggestive of the rupture of a liver abscess into the lung.

Black sputum may be observed in the pulmonary affections of those living in an atmosphere of black dust. This condition is common among coal-miners, and is known as anthracosis. From the inhalation of other mineral dusts, other peculiar sputum-colors may be observed. Thus, those breathing iron-dust have a brownish-black sputum (siderosis); microscopic examination discovers leucocytes and pigment-granules, and the latter give a blue reaction with hydrochloric acid and yellow prussiate of potash.

Bloody Sputum; Hæmoptysis.—Of all varieties of sputum, this is one of the greatest clinical interest. Ordinarily accepted by the laity as evidence of tuberculosis, its relation to other pulmonary troubles has been greatly ignored. In point of fact, very few symptoms present so many clinical relations as does this one.

Hæmoptysis may occur in—

1. Tubercular lung diseases, the special lesions giving rise to it including
    - (a) Collateral congestion around infiltrated areas in the early stages of the diseases. Hæmoptysis from this cause is rarely serious.
    - (b) Rupture of a small aneurysm.
    - (c) Erosion of a vessel. This cause is especially liable to occur during the late stages of tuberculosis.
2. Congestion of the lungs.
    - (a) Passive congestion of organic heart disease, especially of uncompensated mitral lesions.
    - (b) Croupous pneumonia.
    - (c) Hæmorrhagic infarcts.
    - (d) Phthisis.
3. Aneurysm of the aorta rupturing into the respiratory passages. The hæmorrhage in these cases is always extreme, and usually rapidly fatal; not, however, because of loss of blood, but from strangulation.
4. Malignant disease of the lungs; the special characteristics of the symptom being its repeated recurrence.
5. Plastic bronchitis, with detachment of the fibrinous plugs.
6. Gangrene of the lungs, with casting off of sloughs.
7. Ulceration of the larynx, trachea, and bronchi.
8. Blood changes, as in purpura, pernicious anæmia, leucocythæmia, and many of the acute infections.
9. In presumably healthy young persons from unknown causes. This variety is not followed necessarily by untoward symp-

toms; but its occurrence should lead to precautions lest tuberculosis develop.

10. Gouty endarteritis, usually associated with increased vascular pressure, thickening of the radial arteries, and interstitial nephritis. Hæmoptysis from this cause is rarely attended by any serious results.
11. So-called vicarious menstruation. This, as a cause of hæmoptysis, should be mentioned only to be condemned. The associated amenorrhœa is nearly always a conservative symptom. Such cases are nearly always tubercular.

Hæmoptysis is nearly always associated with certain symptoms. Of these, anxiety is one of the most constant. If the quantity of blood lost is large, there will be fainting and vertigo, and even syncope. At the same time it must be remembered that these latter symptoms may result from the mental condition of the patient. Fever is often present, and may assume a high grade. The blood is brought up by the act of coughing, and it is important to remember that the cough serves to perpetuate the bleeding; hence measures should be taken to suppress it.

Hæmoptysis is to be differentiated from hæmatemesis and hæmorrhages from the mouth and pharynx. For the necessary data, the reader is referred to Chapter V. (page 215).

Collection of Sputum for Microscopical Examination.—A number of precautions are necessary in the collection of sputum for microscopical examination, if the results are to be reliable. The vessel in which the expectorated matter is collected should be scrupulously clean, and should be kept covered to prevent the entrance of extraneous materials. As far as possible, the mouth should be cleansed before expectoration, to avoid contamination of the sputum by the contents of that cavity. Specimens obtained shortly after eating should be discarded as liable to contain particles of food. Only those substances which are clearly brought up by coughing should be collected. Finally, the specimen should be brought to the physician in as fresh a state as possible, and he should proceed with its examination without delay. All sputum remaining after making the examination should be thoroughly disinfected with corrosive sublimate, formalin, or carbolic acid. It may then be safely thrown away.

To examine the specimen, a portion of the same is placed on a cover-glass and carefully spread out in as thin and as even a layer as possible. In many examinations this may be all that is necessary. In others, special preparation and staining methods are necessary. These will be explained in the following pages, when referring to the special components of the sputum and their significance.

(*a*) **Red Blood-Corpuscles.**—These are found in small numbers in all sputum. They are not to be regarded as of clinical importance unless the blood is present in sufficient quantity to discolor the sputum microscopically. As a rule, the red blood-corpuscles of the sputum are unaltered. Notable exceptions, however, are found in pneumonia, and in cases of bronchial disease in which the secretions are retained for some time before expectoration.

(*b*) **Leucocytes.**—Leucocytes are found in all specimens of sputum, but are especially numerous in the purulent varieties. Many of them are polymorphonuclear and contain eosinophile granules. Sometimes they contain drops of fat and particles of pigment, and hæmatoidin. In certain of the infectious processes, as croupous pneumonia and tuberculosis, they are found, by means of certain staining processes, to contain micro-organisms.

(*c*) **Epithelium.**—The general impressions among clinical microscopists do not favor the idea that the presence of epithelium of various types affords much practical information. This is directly opposed to what one would *a priori* believe to be the case. Possibly, if the epithelial formations in the sputum were studied systematically, information of considerable value would be obtained.

The epithelium found in the sputum includes large, flat cells, ciliated epithelium, the so-called alveolar cells, and the "heart-disease" cells. The flat epithelial cells of the sputum are derived mainly from the mouth, although they may come from the true vocal cords. Those found in the bloody sputum of hysterical patients are derived from the œsophagus, in which case they appear in large clusters.

To examine the sputum for epithelium, a small quantity should be spread evenly over a cover-glass and treated with acetic acid. This brings the nuclei into view. Or the specimen may be fixed and stained with methylene blue.

Ciliated or columnar epithelium is rarely seen in the sputum, because presenting pronounced changes before it is expectorated. It is sometimes found in the sputum of acute bronchitis, but in such cases it is probably the result of admixture with the nasal discharges. Changed epithelium of this variety may appear in the sputum in the shape of mucus, or goblet-cells in cases of bronchial or tracheal catarrh.

As to the so-called *alveolar cells,* there is considerable discussion as to their nature, some authorities contending that they are, in all probability, changed leucocytes. They are elliptical in shape and contain one nucleus, which, if not already prominent, is made so by the addition of acetic acid. The body of the cells contains numerous pigment-granules, which consist of blood-coloring matter, iron-dust, or carbon. They may also contain myelin and fat-drops. It was claimed by Buhl that these cells are characteristic of what he is pleased to call desquamative pneumonia. Troup regards them as evidence of an obstinate catarrh of the apex, and believes, too, that they appear before other physical signs are in evidence. According to von Jaksch, they are also found in pneumonia, chronic bronchitis, and pulmonary tuberculosis. Vierordt thinks "it quite probable that they are mostly or altogether made up of white blood-corpuscles, enlarged by metamorphosis of their protoplasm and partly by absorption of small particles. In part, also, they may come from the deeper layer of the bronchial epithelium."

The "heart-disease cells" consist of alveolar epithelium larger in size than those above described, and containing numerous granules of brown, or golden-yellow, pigment. They are said to indicate brown induration of the lungs. Their epithelial origin has also been contested, von Noorden claiming them to be "pigment-bearing eosinophile leucocytes." Their relation to brown induration of the lungs has not, however, been disputed.

(*d*) **Elastic Fibres.**—The presence of elastic fibres in the sputum is a symptom of great clinical importance, as it indicates destruction of lung-tissue. The diseases in which elastic fibres occur in the sputum include pulmonary tuberculosis, bronchiectasia, pulmonary abscess and gangrene of the lungs, and pneumonia. In the latter disease the appearance of the elastic fibres may constitute the sole evidence of pulmonary destruction, the disease subsequently undergoing a favorable course. It is to be presumed in such cases that the destruction of tissue was limited. In pulmonary gangrene the quantity of elastic fibres is not in consonance with the amount of tissue-destruction. This fact was explained by Traube: that they are destroyed before expectoration by ferments formed in the gangrenous process. When examining sputum for elastic fibres, it is of the greatest importance that it be obtained pure, for it may be that particles of food may be mistaken for them.

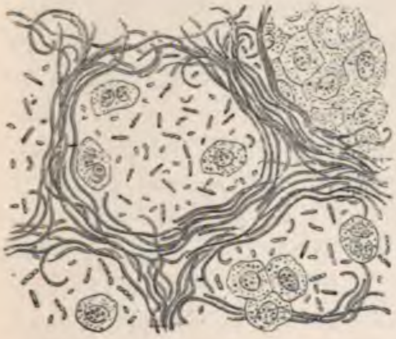
For the detection of elastic fibres in the sputum the method proposed by Sir Andrew Clark, as modified by Osler, is the best. A small quantity of the suspected purulent sputum is pressed out between two glass plates, on a black background, the lower being six inches square, the upper but four inches. The elastic fibres appear as grayish-yellow spots on the black background, and may be examined *in situ* by the microscope, using an objective of low power. Or the suspected pieces may be removed and mounted on a slide. As viewed by the microscope, they appear as branching fibres having a double outline. When derived from the lungs, they present an alveolar arrangement. When derived from the bronchial tubes, they are branched.

When the elastic fibres are few in number, a large quantity of the sputum should be mixed with an equal amount of a 10 per cent. solution of caustic potash and boiled. The jelly-like mass thus formed should be diluted with water and permitted to stand for twenty-four hours. The elastic fibres settle to the bottom, whence they may be collected and examined microscopically. Or sedimentation may be effected by the centrifuge.

(*e*) **Fibrinous Coagula.**—Fibrinous coagula may appear in the sputum of fibrinous bronchitis and croupous pneumonia, and take the shape of the bronchioles and their ramifications, in which they are formed. Those expectorated in croupous pneumonia are usually quite small. Their presence in this disease indicates a severe type. In fibrinous bronchitis they present a high degree of development, and cases have been reported in which the main trunk was a moderately large tube, to which the branches thereof, to the minutest ramifications, were attached. In fact, the presence of such remarkable moulds of the bronchial tubes constitutes the diagnostic symptom of fibrinous bronchitis.

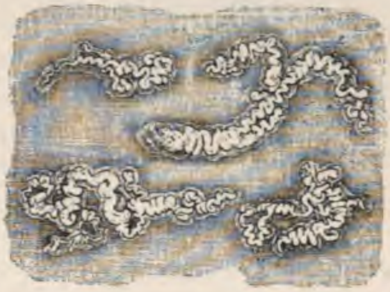
Ordinarily, fibrinous coagula appear in the sputum as small lumps mixed with mucus. When shaken gently in water, they unravel and display their true character. They are found to be grayish-white in color, and hollow. A transverse section usually shows that they are made up of several layers.

FIG. 92.



Elastic fibres in the sputum.—After von Jaksch.

FIG. 93.



Spirals from the sputum in a case of bronchial asthma.—After von Jaksch.

Larger casts derived from the trachea and bronchial tubes may be observed in croupous inflammations of these structures.

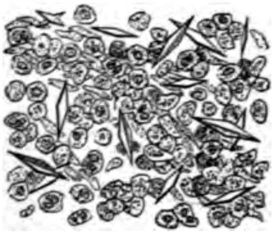
(*f*) **Spirals.**—These are spiral-shaped bodies containing a central thread, found for the most part in the sputum of bronchial asthma, and also in other affections having in common a thick gelatinous sputum and pronounced dyspnœa. The central thread is spoken of as the central fibre, while the spiral mass about it is known as the mantel. With care and experience these spiral bodies may be seen by the unaided eye, appearing as spiral fibres with a wavy, shining stripe within. Under the microscope, the mantel is seen to consist of loose, corkscrew-like fibres wound about the shining central thread. Some of the spirals consist of mantel alone, while others are limited to the nude central fibres wavy in outline.

These spiral bodies are generally regarded as highly diagnostic of bronchial asthma. They are especially numerous in the expectoration at the end of the seizure. They are also found in various types of chronic bronchitis, emphysema, the bronchitis of heart disease, croupous pneumonia, pulmonary tuberculosis, and hæmorrhagic infarctions.

Gerlach has succeeded, by twisting particles of mucus, in producing these spirals with their central threads. This has led to the suggestion that they originate in the bronchial tubes by a twisting process. The mechanism of this is, however, not at all understood.

(*g*) **Charcot-Leyden crystals** are intimately associated with the spirals. They consist of shining blue elongated octahedra of various sizes. The points where these crystals are found appear to the naked eye as dry crumbs. For microscopic investigation a low-power objective is required. They are regarded as very important diagnostic indications of bronchial asthma. Examination of the sputum freshly expectorated at the beginning of the attacks shows these crystals to be absent. But if the specimen is kept covered for twenty-four hours to prevent evaporation, crystals will be seen on the spirals. This fact seems to favor the idea that they are formed from the constituents of the spirals. In the sputum discharged later in the asthmatic seizure, the crystals are found in abundance in the parts occupied by the spiral formations, but not elsewhere. The Charcot-Leyden crystals are found also in acute bronchitis, chronic croupous bronchitis, and tuberculosis, but not with the regularity noticed in connection with bronchial asthma.

FIG. 94.



Charcot-Leyden crystals from the sputum of an asthmatic patient.—After von Jaksch.

(*h*) **Corpora amylacea** or **starch corpuscles** are small bodies found in the sputum of hæmoptysis and pulmonary gangrene, the significance of which is not understood. They are partly round and partly angular in shape, and give the amyloid reaction to iodine.

(*i*) **Connective tissue** is rarely found in the sputum. Its presence is significant of a destructive process in some portion of the respiratory tract.

(*j*) **Tubercle-bacilli.**—The examination of the sputum of suspected cases of pulmonary tuberculosis for the detection of these micro-organisms is now regarded as important, as a routine measure, as is the physical exploration of the chest. Advanced technique makes it possible to discover their presence in a very few minutes. The reagents necessary are a carbol-fuchsin solution and an acid methylene-blue stain.

The formula for the carbol-fuchsin solution is as follows:

Fuchsin, ...	1 gramme.
Absolute alcohol, ...	10 c.c.
Five per cent. carbolic acid solution, ...	100 c.c.

The acid-blue solution is prepared according to the following:

Methylene blue,	2 grammes.
Sulphuric acid,	25 grammes.
Distilled water,	75 c.c.

A portion of the suspected specimen of sputum is placed between two thin cover-glasses and spread in a thin layer by compression between the fingers. The bacilli are found most numerous in the small, cheesy-looking particles so common in tuberculous sputum; hence such portions of the sputum should be selected for investigation. Care must be observed not to place too much sputum on the cover-glass, for in that case, compression is liable to force all of it out. The cover-glasses must be scrupulously clean; hence they should be washed first with water, and then with absolute alcohol.

The carefully-spread cover-glasses are next separated. One of them is selected for examination. The sputum on it is carefully fixed by drying over an alcohol flame, taking due care not to char the specimen. This accident is less likely to happen if the physician holds the glass in his fingers than if he uses forceps for the purpose. Next the carbol-fuchsin solution is dropped on the surface of the film until the latter is covered evenly with the stain. Then, holding the glass in forceps, it is placed over the flame until it is observed to steam, care being taken that the heat is not maintained until boiling takes place. It is then removed, and as soon as the steaming ceases it is replaced over the flame. This procedure is gone over six or seven times. The stain is next washed off in a gentle stream of water. The specimen is now ready for the acid-blue solution, which is applied drop by drop until the cover-glass is covered with the same. It is permitted to remain a couple of minutes, when it, in turn, is washed off under the spigot.

The carbol-fuchsin solution stains the entire specimen red. The acid in the methylene-blue solution decolorizes all the specimen excepting the tubercle bacilli. The methylene blue stains all the decolorized structures blue. The specimen is now ready for mounting. If there is no desire to make a permanent mount, the cover-glass may be placed on a slide while still wet. The tubercle bacilli appear as minute red rods, all other objects in the field being stained blue. No other micro-organism stains in this way excepting the bacillus of leprosy, and that is not likely to cut any figure in producing diagnostic errors when searching for tubercle bacilli.

When tubercle bacilli are few in number, the procedure proposed by Biedert may be adopted. One ounce of the suspected sputum is mixed with an equal quantity of distilled water, and from four to eight drops of liquor potassæ solution added. The mixture is then boiled. It is then placed aside in a conical glass for twenty-four to forty-eight hours (but not more), to settle. A portion of the sediment thus formed is to be treated as above described and examined for the bacilli. The procedure may be hastened by securing sedimentation by means of the centrifuge.

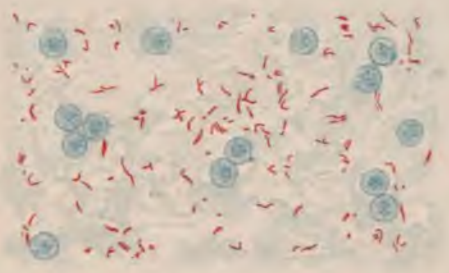
The tubercle bacilli are found to be small, slightly curved, rod-like bodies, ranging in length from 1.5 to 3.5 μ. They sometimes contain spore formations, which do not stain by the processes advocated for the bacilli themselves. Hence they appear under the microscope as small breaks in the continuity of the bacillus.

The presence of tubercle bacilli in the sputum is positive proof of the existence of tuberculosis of the air-passages. They may be present even when the disease has not advanced sufficiently to produce the physical signs of tuberculosis. Negative results are to be taken with some reserve.

(*k*) **Micro-Organisms of Pneumonia.**—Three micro-organisms have been described as having a specific relation to croupous pneumonia. These include (1) Friedländer's pneumococcus; (2) Fränkel's pneumococcus; and (3) Pio Foa's coccus. Friedländer's pneumococcus, although not the first aspirant for favor as the disease-producing factor of pneumonia, was the first to which much importance was attached. It was described by its discoverer as an oval body 1 μ in length, occurring in pairs or in chains, and enclosed in a capsule. Fränkel's pneumococcus, like that of Friedländer's, also occurred in pairs, and had a capsule; it was oval or lancet-shaped. Weichselbaum's bacteriological researches of 129 cases of pneumonia showed Fränkel's coccus in 64, and Friedländer's in but 9. Other cases exhibited streptococci and staphylococci. It would thus seem that the subject is one requiring considerable investigation before any positive conclusions can be reached. One difficulty heretofore is that bacteriologists have made but little effort to differentiate the types of pneumonia from which the several micro-organisms were derived. Thus far it has been decided that the micro-organisms in question do bear some relation to croupous pneumonia, but exactly what, cannot be stated. None of them answer the tests tabulated by Koch as establishing the pathogenetic character of a micro-organism. They are also found in the healthy secretions of the mouth; hence it is fair to presume that there must be an additional factor required to make them active. At the present time, Friedländer's pneumococcus is the only one accepted seriously as having an important relation to pneumonia. It may be demonstrated by Gram's method of staining; or the following, advocated by the discoverer and quoted by von Jaksch, may be used:

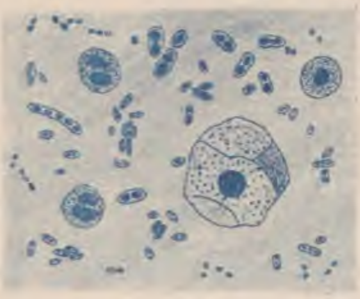
The cover-glass preparation is passed three times through the flame of a Bunsen burner, placed for a minute or so in a 1 per cent. solution of acetic acid, the fluid removed by blowing upon it through a pointed glass tube, the preparation dried in the air and placed for a few minutes in a saturated aniline water and gentian-violet solution, finally washed with

FIG. 95.



Tubercle-Bacilli, stained by the Ziehl-Neelsen method.

FIG. 96.



Pneumococci.

water, and mounted on a slide for examination. When this is done, a number of capsulated rod-like micrococci may be seen with the microscope.

(*l*) **Bacillus of Influenza; Pfeffer's Bacillus.**—This is a small bacillus with rounded extremities, staining more deeply at the ends than at the middle. They are found in large numbers in the sputum of influenza. The cover-glass preparation is to be stained with aniline-oil, gentian-violet solution, somewhat decolorized with alcohol, and counter-stained with aqueous solution of aurantia.

(*m*) **Actinomyces.**—This fungus may occasionally be observed in the sputum. To the unaided eye it appears as small yellowish-white or greenish-yellow masses, the size of a millet seed. For microscopic examination no staining is necessary, as the rounded, club-shaped bodies, projecting in all directions from a central mass, are readily recognized.

(*n*) **Echinococcus.**—Echinococcus cysts are of occasional occurrence in the lungs. Under such circumstances, the characteristic hooklets and pieces of the membrane may be found in the sputum.

Pain as a Symptom of Thoracic Disease.—Pain is not, as a rule, a prominent feature of disease of the respiratory apparatus unless the pleuræ are involved. In acute and chronic catarrhal bronchitis, and indeed in bronchial affections generally, any subjective sensations consist of sensations of burning, rawness, stuffiness, etc. When there is great suffering, it is from discomfort rather than from actual pain. Involvement of the pleuræ in any of the respiratory pathological processes brings with it an incidence of pain, often severe. Thus we find pain not uncommon in the early stages of tuberculosis and in croupous pneumonia. The source of pain in the latter affection is not usually attributed to the pleura; nevertheless that membrane is commonly inflamed to a considerable extent in this disease,—sufficiently so, indeed, to account for the pain which happens to be present.

Pleurisy, however, is the disease of the chest *par excellence* associated with pain. Early in the course of the affection a severe stitching pain is experienced, and this is aggravated on the slightest motion of the chest. It leads to restrained breathing, and interferes with the act of coughing. As soon, however, as effusion sets in, and the inflamed surfaces of pleura are no longer in contact, the pain ceases, or at least is greatly ameliorated.

In general practice, the most frequently observed causes of pain in the chest are intercostal neuralgia and intercostal rheumatism. These affections are to be differentiated by reason of the fact that rheumatism (myalgia) is aggravated by pressure or by movement of the affected muscle. Neuralgia is not so aggravated, and is associated with tender points. At the same time, there is important negative evidence in the absence of the physical signs pointing to inflammation of the pleura.

Respecting intercostal neuralgia, its frequent association with herpes should be borne in mind. The diagnosis in such cases is made by the discovery of groups of herpetic vesicles following the course of an intercostal nerve, or the history that such an eruption has recently been present. Neuralgia from herpes zoster is at any time very obstinate to treatment.

Whooping-Cough.

Whooping-cough is one of the infectious diseases, the nature of the infecting organism, however, being unknown. It has been determined as a matter of fact that children, especially those who have not reached their sixth year, are especially liable to it. The contagiousness of the disease is greater during the earlier weeks, *i.e.*, before the appearance of the characteristic whoop.

Whooping-cough is ushered in by a cough which differs in no particular from those arising from an ordinary cold. Unlike the latter, the cough does not yield to treatment, becomes more and more annoying, and *exhibits remarkable nocturnal exacerbations. An examination of the chest fails to discover physical signs in proportion to the severity of the symptoms.* These two points may be regarded as strongly suggestive, diagnostically, even before the appearance of the characteristic whoop. After a few weeks the distinguishing whoop is manifested, and then the diagnosis is clear. This phenomenon shows itself as a long-drawn crowing inspiration succeeding a number of violent expulsive coughing acts. By the time the whoop appears the patient is well-nigh breathless, as evidenced by his purplish or cyanotic countenance and exhaustion. The distress occasioned by the paroxysms is evident to all. The little sufferer endeavors to restrain them as far as it is in his power to do so. It is not infrequent for the paroxysms of coughing to terminate in vomiting.

The frequency and severity of the paroxysms vary greatly in different cases. They may, however, be mitigated by careful attention and treatment. After a few weeks the spasmodic stage declines, and the case progresses uninterruptedly towards recovery.

Whooping-cough is a disease with a number of complications and sequelæ. The violent paroxysms sometimes provoke hæmorrhagic extravasations. The most frequently observed of these are subconjunctival ; but they may take place into the internal viscera, producing serious symptoms. The *vomiting* is ordinarily a symptom of little consequence, but at times it may be so persistent and continuous as to endanger life. *Nervous complications* include laryngeal spasm, which in very young subjects is especially dangerous. As evidence of the remarkable nervous irritability of the patient, may be noted the not infrequent occurrence of convulsions. In many instances these are neurotic only ; but in a large proportion they are the result of serious organic lesions, as hæmorrhage, thrombosis, and pulmonary collapse. Those cases dependent upon cerebral hæmorrhage and thrombosis are, of course, attended by the characteristic symptoms of these lesions.

The pulmonary complications include collapse of the lungs and broncho-pneumonia. The former is especially liable to occur in infants; the latter in patients who have reached or passed the second year of life. Both are serious.

Whooping-cough seems to have the ability of awakening into activity any dormant constitutional defect. Hence it is not unusual for tubercular disease to follow closely upon it. The same may be said of hereditary syphilis and nervous instability.

CHAPTER XIII.

THE HEART.

As a matter of routine, the heart should be made the subject of thorough physical examination in all cases of acute disease. The thoroughness with which it does its work is a fair index of the ability of the patient to withstand the ravages of disease. Ordinarily, sufficient information may be derived from the pulse, details concerning which have been referred to in a preceding chapter. The fact that the pulse gives valuable information affords no reason for neglect of a careful investigation of the condition of the heart itself. In chronic diseases cardiac changes are not uncommon, especially in connection with affections of the kidneys, lithæmia, and degenerative changes in general. In cases in which the heart itself is the seat of disease, the necessity for examination goes without saying. Such cases are suggested by breathlessness on slight exertion, œdema and pulse-changes, and other symptoms.

The methods employed for the physical examination of the heart are—

1. Inspection.
2. Palpation.
3. Percussion.
4. Auscultation.

1 and 2. **Inspection and Palpation.**—These methods of examination as applied to the heart may well be studied together, as each gives the same information of which the other is capable, and one supplements or confirms the observations obtained by the other. Palpation may be regarded as giving the more positive results.

In the practice of inspection and palpation as related to the condition of the heart, as in everything else, systematic method of examination is essential to accuracy. It is important to bear in mind that more mistakes are made from deficient observation than from ignorance. Attention should be directed to the following points:

a. Alterations in the color of the chest and neck, head and arms.
b. The prominence of the veins in the localities just mentioned.
c. The location and character of the apex-beat.
d. The location and character of pulsations other than those of the apex-beat.
e. The contour of the chest over the heart.

f. Other abnormal prominences, including bulging of the intercostal spaces.

(*a*) **Alterations in the Color of the Chest, Neck, Head, and Arms.**—A prominent feature of many cases of cardiac disease with failing compensation is a general cyanosis, consideration of which will be found elsewhere. Aside from this may be occasionally observed a localized venous congestion, the significance of which will vary according to its location and mode of onset. An intense venous congestion of the upper portion of the body coming on suddenly may be regarded as suggestive of that rare condition, the rupture of an aortic aneurysm into the superior vena cava. It is usually associated with œdema of the affected parts. The congestion and œdema are sometimes limited to the head and arms, in which case the obstruction must be situated above the azygos vein. If the chest participates in these appearances, then we know that the obstruction must be below the point of entrance of the azygos vein into the superior vena cava. It is of interest to note that in these cases with lividity of the chest, there is a sharp line of demarcation between the congested and unaffected parts.

The changes above mentioned may undergo some modification by reason of certain attendant circumstances. If, for example, the obstruction has preceded the rupture for a long time, and there has been opportunity for establishing a collateral circulation, the blood-current is not interfered with equally on the two sides. Thus, in the case of a long-standing pressure exerted upon the left innominate vein, the rupture of the aneurysm produces œdema of the right side only. Again, if the rupture takes place in such a position as to direct the blood into the right innominate vein, the œdema and lividity will be confined to the right arm.

The lividity and œdema may also be unilateral in cases of rupture of an aneurysm into one of the innominate veins, the symptoms being limited to the upper portion of the body on the corresponding side.

Aneurysm of the aorta is not the only form of tumor which may produce intrathoracic vascular obstruction, with resulting disturbance of the tributary circulation. Indeed, solid tumors may exert a more prominent influence in this respect, and are more frequently observed in practice as the cause of these circulatory disturbances. Very often, however, they grow so gradually as to give abundant opportunity for the establishment of a collateral circulation. For example, a bunch of veins extending from the corresponding axilla to the groin may be observed.

(*b*) **Prominence of the Veins of the Neck, Thorax and Arms.**—Undue and persistent fullness of the veins may be taken as evidence of distention and weakness of the right heart. This condition is often observed as a temporary condition, as the result of violent exertion, coughing during attacks of bronchitis, straining at stool, etc. Account must always be taken of the normal standard of the individual, as the veins vary in prominence according to the patient's degree of stoutness or leanness.

A band of superficial venules is sometimes observed as a belt around the lower portion of the thorax at the level of the ensiform cartilage, and a little above the costal margin. Its exact significance is unknown, for it has been observed in persons who are, so far as could be ascertained, in the best of health.

When venous distention has existed for any length of time, the valves of the affected vessels become knotted, owing to the strain, and may eventually become impaired. Especial attention should be paid, when examining the veins of the neck, to the point at which the jugulars enter the subclavians, as here may be formed quite a pouch, when the venous distention is extreme.

The subject of venous pulsation has already been considered, and need not be mentioned at this time. (See Chapter III.)

(*c*) **The Location and Character of the Apex-Beat.**—The normal position of the apex-beat in the healthy adult is in the fifth interspace, and, to use the expression generally employed in this connection, well within the nipple-line. For scientific accuracy, the nipple should hardly be used as a landmark for locating the apex, as in women it is most decidedly an uncertain point of location, varying, as it does, with the size and consistency of the breast. In men, it is a fairly fixed point, in them varying but little. It is far better, however, in all cases, to designate the distance at which the apex-beat is felt from the mid-sternum. This varies from two and a half to three inches. In children, prior to the age of six years, the apex-beat is found in the fourth interspace, and farther from the median line than in the adult. In the aged, it is not unusual to find it in the sixth or even in the seventh interspace, and nearer the mid-sternal line than above specified.

One must not, however, be too strict in maintaining the fifth interspace and two-and-a-half to three inches from mid-sternum as *the* normal position for the apex-beat. It may change with the position of the body, gravitating to the left with the left lateral decubitus, and *vice versa.* During full inspiration it is displaced downward and to the right, and it may be absent entirely when the lungs are fully distended.

Strange as it may seem, recognition of the different interspaces is not always correct. Mistakes arise from confusing the space between the clavicle and the first rib for the first interspace. To be warned against the possibility of this error is sufficient to save the observer from making it. He will be surprised to find the comparatively great distance between the second interspace and the clavicle. To make sure that an accurate counting of the interspaces has been made, it may be well to bear in mind that a line drawn through the base of the ensiform cartilage strikes the lower portion of the fifth interspace.

Departures of the Apex-Beat from the Normal Standard.—The apex-beat may be altered in quality or force, or it may be displaced from its normal situation.

Alterations in Quality or Force.—The normal apex-beat occupies a space so small that it can be readily covered by the end of one's thumb. It is larger when the patient leans forward, when the chest-walls are thin, and at the end of the act of expiration. The shape of the thorax may influence the position of the beat. Thus, in persons with a short, wide thorax it may be found in the fourth interspace, while in those with a long, narrow thorax it may be felt in the sixth interspace.

In the majority of healthy individuals the apex-beat may be perceived by inspection. A good light and proper position are necessary. The observer should have his eye on about the level of the beat, and should view it in profile. The discovery of the beat by palpation does not require as much care.

Sometimes the apex-beat is not detectable at first by either inspection or palpation. This does not signify an abnormal condition, unless it be associated with an abnormal weakness of the pulse. It may be due to unusual separation of the heart from the thoracic walls, or to undue thickness of the latter by reason of large deposits of adipose. In case the pulse is found to be of normal strength, it is fair to presume that the heart is acting with its wonted force. In order to bring the apex-beat within the perceptive powers of the sight or touch, especially the latter, let the patient lean somewhat forward.

Pathologically, the apex-beat may fail of perception because of weakness of the cardiac impulse, or of separation of the heart from the thoracic walls by pericardial effusion or an emphysematous lung. Sometimes it is due to smallness and weakness of the heart, as in the cardiac atrophy of old age. When the absence is the result of pericardial effusion, it will be found that it usually becomes manifest when the patient bends forward. At the same time it will be found to be pushed upwards, and will occupy the fourth interspace. The intercostal spaces may also be found bulging.

In the case of emphysema, not only may the apex-beat be apparently weakened by separation from the chest-wall, but there may be an actual diminution in force because of the interference of the expanded lung with the muscular action of the heart.

Having discovered the position of the apex-beat, it is a good practice to mark its position with the dermatographic pencil.

By reason of pathological conditions, *the apex-beat may be displaced upwards, downwards, outwards* or *inwards.*

Displacement of the apex-beat to the right side of the chest may be found in that rare condition, transposition of the viscera, in which case the liver will be found to occupy the left hypochondrium. Very exceptionally (only two such cases have been reported), the heart will be found to be the only viscus transposed, all the other organs occupying their normal places.

Pathologically, displacement in the same direction may result from large accumulation of fluid in the left pleural sac, in extreme cases the apex-beat being found outside the right nipple-line. Displacement to the right, though in less degree, may result from accumulation of air in the left pleural sac, tumors, enlargements of the left lung, and retraction of the right lung.

Displacement of the apex-beat to the left may result from right pleural effusion, right pneumothorax, right pleural tumors, tumors of the right lung, and retraction of the left lung.

Upward displacement of the apex-beat may occur in consequence of pressure exerted upon the heart from below, *e.g.*, abdominal tympanites, ascites, enlargement of the liver and spleen, and abdominal tumors generally. In cases of enlarged liver the displacement is usually upwards and to the left; in case of enlarged spleen, upwards and to the right.

Downward displacement of the apex-beat, even to the seventh or eighth interspace, is found in connection with hypertrophy and dilatation of the heart. At the same time, when this is the cause of the displacement, the apex may be found outwards, even as far as the axillary or mid-axillary line. Hypertrophy and dilatation are differentiated by the attendant symptoms and the character of the cardiac impulse. In the case of dilatation, there will be the usual phenomena of failing compensation, and there will be found a widely-distributed heaving movement over the præcordia. In the case of hypertrophy, the cardiac impulse will be strong, prolonged, and well-defined.

The heart may be displaced downwards by any influence exerting pressure from above or lack of support below. Hence it may be a result of solid or aneurysmal tumors, pulmonary emphysema, and descent of the diaphragm consequent upon collapse of the abdominal viscera.

Upward displacement of what is apparently the apex-beat occurs in pericarditis with effusion. I say "apparently what is the apex-beat," because it is contended by some that there is no displacement of the apex-beat, but that the pulsation seen in the third or fourth interspace in cases of pericardial effusion is produced by the right auricle and the conus arteriosus acting on the chest-wall.

As already stated, the impulse of the normal heart is limited to a small area about the apex-beat. This area may be increased by causes which lessen the amount of lung covering the heart, as phthisis, and pleural adhesions; by tumors, which crowd the heart against the thoracic wall; by pericarditis with small effusion, in which case the impulse is a diffuse wave occupying the second to the fourth interspaces; by hypertrophy and dilatation of the heart.

(*d*) **The Character and Location of Abnormal Pulsations.**—In the clinical study of pulsations about the chest, it is of the greatest importance that their relation to the systole of the heart be carefully noted.

Epigastric pulsation is observed with greater frequency than pulsation in any other locality. It often serves as the foundation for an incorrect diagnosis of aneurysm, when, in point of fact, aneurysm is the last condition to think of in the presence of this symptom. The causes of epigastric pulsation exist in quite a variety. The majority of cases are observed in neurotic subjects, who are anæmic and dyspeptic. There is in all probability some vaso-motor disturbance, the tonus of the vessel being relaxed.

It may be present as the result of downward displacement of the heart. It is then due to contractions of the right ventricle. Sometimes this pulsation is communicated to the adjacent portion of the liver.

Epigastric pulsation is sometimes due to the transmissions of the pulsations of the abdominal aorta through an overlying solid tumor. In such cases the pulsation will not be expansile, as in aneurysm, and the pulsations will disappear when the patient is on his hands and knees, as in that position the weight of the growth removes it from its contact with the aorta.

Epigastric pulsation from aneurysm is expansile; it is not affected by posture; there is the characteristic aneurysmal murmur; and the pulsations are apt to be strong during expiration and weak or absent during inspiration.

Pulsation in the second left interspace is derived from the pulmonary artery or dilatation of the conus arteriosus. It may result from dilatation of the pulmonary artery, or from conditions in which that vessel approaches nearer than the normal to the chest-wall. The latter occurs in cases of retraction of the lung; hence pulsation in the second left interspace suggests a thorough examination of the state of the lungs.

Pulsation of the right auricle is observed to the right of the sternum. It may be observed in cases of dilatation of that portion of the heart. The movements are undulatory, owing to the contractions of the ventricles following those of the auricle.

Pulsation of the left auricle is never observed excepting in children, and then only in cases of marked hypertrophy. The pulsation is located in the second to the third left interspaces, a short distance from the edge of the sternum. It is presystolic in time.

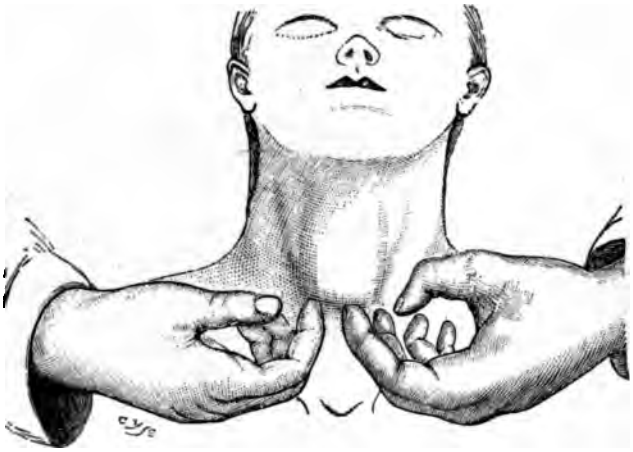
In these cases stenosis of the left auriculo-ventricular orifice will also be found.

Pulsations communicated to the upper portion of the sternum are, for the most part, the result of aortic aneurysm. The sensation gives to the hand one of heaving and expansion. It may be associated with a thrill.

Diastolic shocks may be discovered by palpation. In the case of sacculated aneurysm of the ascending or transverse portion of the aorta it is felt near the base of the heart. It results from the falling back of an unusually large column of blood.

Tracheal Tugging.—This is a symptom found in connection with aneurysm of the aorta. It is to be elicited as follows: "Place the patient in the erect position, and direct him to close his mouth and elevate his chin to the fullest extent; then grasp the cricoid cartilage between the finger and thumb and exert gentle upward pressure. If aneurysm exist, the pulsations of the aorta will be distinctly transmitted through the trachea to the fingers. The act of examination will increase laryngeal distress should this accompany the disease." (Oliver.) Ewart suggests that the cricoid cartilages be elevated by the tips of both index fingers. Otherwise he advises proceeding in the same way as does Oliver. This symptom is only present when the aneurysm is situated so as to press upon the left bronchus or the adjacent portion of the trachea.

FIG. 97.



Dr. Ewart's plan of eliciting tracheal tugging. The observer is supposed to be standing behind the patient.—After Sansom.

Valve Shocks.—Musser is authority for the statement that the shocks produced by closure of the valves are discernible by palpation. It seems almost incomprehensible that such should be the case. It is necessary, in order to detect them, that the hand be placed evenly over the præcordial region. It is essential that the chest-walls be thin. The shock communicated by the aortic and pulmonary valves is especially liable to be present in cases in which intravascular tension is high, the shock following the impulse of the heart. The mitral and pulmonary valves are less likely to communicate this sensation. It is felt in the left fourth interspace near the sternum, is systolic in time, and is associated with dilatation of the heart.

Thrills.—Thrills may be either pericardial or endocardial. The former are produced by the rubbing of roughened pericardial surfaces against each other; the latter by the production of the vibration of the blood passing over a roughened surface. In the clinical study of thrills, it is important that their location and relation to the cardiac cycle be carefully noted.

A systolic thrill felt in the second right intercostal interspace near the border of the sternum suggests aortic aneurysm or stenosis of the aortic orifice.

A systolic thrill felt over the pulmonary area indicates obstruction at the pulmonary orifice.

A diastolic thrill felt over the pulmonary artery nearly always indicates aortic regurgitation, and is associated with the water-hammer pulse.

A presystolic thrill observed best at the apex is a sign of mitral stenosis. Careful observation shows that it terminates at the moment that the apex-beat is felt. It is the most frequently encountered of all thrills.

A systolic thrill in the above-mentioned situation means mitral regurgitation. It is always associated with a mitral systolic murmur, and is gradual in its termination.

A systolic thrill felt best at the lower end of the sternum may accompany tricuspid regurgitation.

Pericardial thrills are frequently described as "*friction fremitus.*" They are present during both systole and diastole, and impart a sensation to the examiner of being near the surface. Usually it is detected by light pressure, though sometimes it is necessary to palpate deeply in the intercostal spaces, especially the third and fourth.

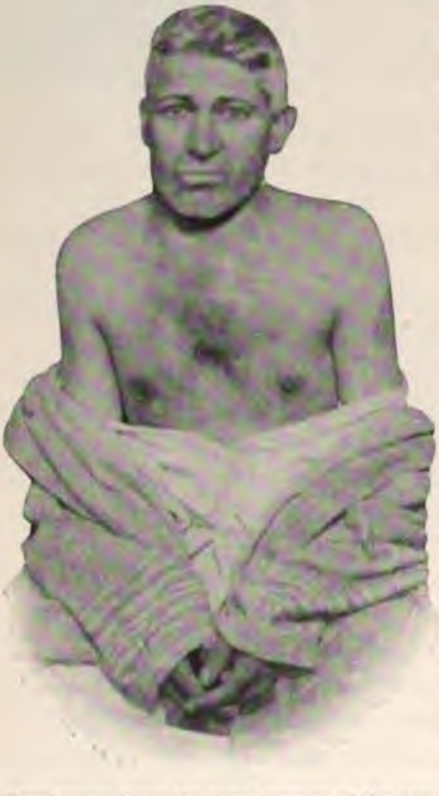
All thrills are felt best with the patient sitting upright or bent slightly forwards.

(*e*) **The Contour of the Chest Over the Heart.**—Ordinarily, the præcordia presents no difference from the corresponding portion of the right side of the chest. Even in the presence of disease in adults it is rare, indeed, that there is any bulging of the præcordia. But in children, as a result of cardiac hypertrophy or dilatation, this portion of the thorax may be unduly prominent, and this condition may persist throughout life, although not to the extent one would expect from the appearances presented in the early days of the lesions. Pericardial effusion in children rarely gives rise to this condition, its alterations in the thoracic walls being limited to marked bulging of the intercostal spaces.

Local bulgings are sometimes observed as the result of aneurysms which, by pressure, have eroded the overlying ribs. In such cases, the characteristic expansile pulsation is associated with the prominence. A bulging localized to the apex of the heart occurs sometimes in aneurysm of that organ.

A retraction or sinking-in of the præcordia is sometimes the result of an old pericarditis. It is a rare result of this, however. It occurs more frequently following empyema.

FIG. 98.



Prominence about the upper portion of the sternum due to aortic aneurysm.

Retraction of the intercostal spaces overlying the heart with the apex-beat occurs as a result of pericardial adhesions.

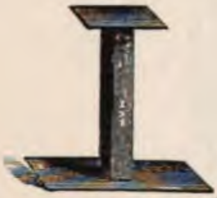
(*f*) **Localized bulgings** may also be the result of solid tumors. They are very rare symptoms.

3. **Percussion.**—The practice of percussion in diseases of the heart involves the same attention to details as described in the chapter on the Physical Examination of Respiratory Diseases; hence they need not be repeated here. It is well, however, to speak of the great value of the Sansom pleximeter in the detection of fine differences in percussion-sounds, as enabling one to delimit areas of relative dullness with an accuracy not possible with the unaided fingers. I might say, in this connection, that there is hardly a problem in physical diagnosis concerning which more mistakes are made—even by masters in the art of percussion—than the outlining of the deep cardiac dullness. This statement is well borne out by the discrepancies between the results afforded by percussion and those of the X-ray in the investigations made at the Massachusetts General and Boston City Hospitals by Williams and others.

Sansom's Pleximeter.—The pleximeter devised by Sansom offers certain advantages possessed by no other instrument. "A thin, flat, oblong plate, of an inch by an inch and a half, has on its upper surface a slender vertical column, an inch and a half in height, which is surmounted by a smaller plate, three-fourths by three-eighths of an inch, set parallel to the former plate. The little instrument is held by the fore- and middle-fingers of the left hand applied on each side of the vertical column, the sensitive tips of the fingers resting on the upper surface of the larger horizontal plate. The lower surface of this latter is applied closely to the wall of the chest, and percussion by one or two fingers of the right hand, with an even and not too forcible stroke from the wrist, is made upon the upper plate. It is obvious that the whole of the pleximeter vibrates with the stroke.

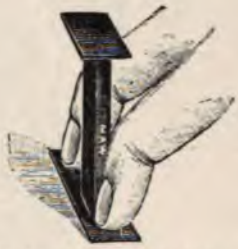
"It is quite unnecessary, in the description of the use of this little instrument, to enter upon the refinement of acoustics; it is better for the observer to think not in terms of sounds, but rather in terms of vibrations. These vibrations are appreciated both by the ear and by the tactile sense. The little instrument is so constructed that vibrations are communicated directly to the organs best fitted to estimate them—the tips of the fingers. Let the mind of the observer be fixed on vibrations; whether these be sonorous and appreciated by the ear, or mechanical and appreciated by the tactile sense, are points of but secondary importance."

FIG. 99.



Sansom's pleximeter.

FIG. 100.



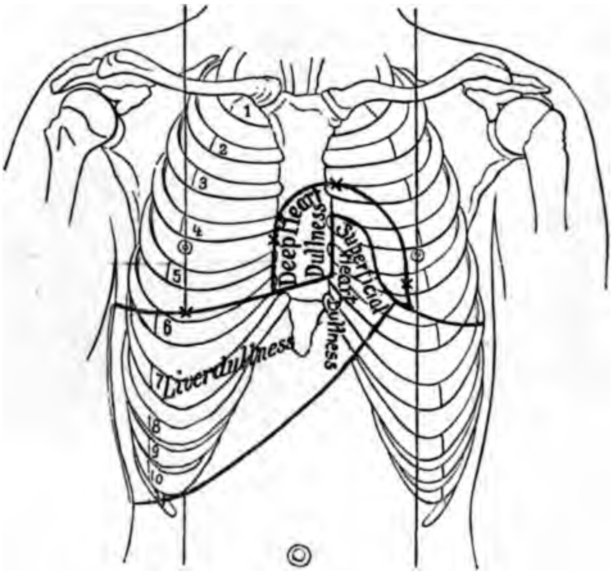
Method of using Sansom's pleximeter.

Anatomical Relations of the Heart.—The heart occupies a position beneath and to the left of the sternum. A certain portion of the organ lies in direct contact with the anterior thoracic wall, the remainder being covered by projecting portions of the lungs. It is important to bear in mind that it is the right side of the heart which lies anteriorly. Only a narrow margin of the left side is viewed from in front at the autopsy when the heart is normal in size and position.

Superficial or Absolute Cardiac Dullness.—Percussion of the portion of the thorax against which the heart directly lies gives absolute dullness. Hence it has been termed the area of absolute or superficial dullness. It includes a triangular space bounded by lines connecting one point at the mid-sternal line opposite the fourth costal articulation, a second in the mid-sternal line opposite the sixth costal articulation, and a third over the apex-beat. This area is very readily defined by percussion.

Area of Relative or Deep Cardiac Dullness.—The overlapping of the heart by the contiguous free borders of the lungs necessarily obscures the dullness which unavoidably appears on percussion over a solid organ. At the same time, it must be remembered that the margin of overlapping lung is comparatively thin, and strong percussion-blows must bring out the physical characteristics of the organ beneath. It will thus be appreciated that the area of deep cardiac dullness must correspond pretty closely with the actual size of the heart. Its limits are greater than those of superficial dullness. Its upper boundary will be found at about the third cartilage; its left at the nipple-line; its right projects slightly beyond the line of absolute dullness,—say to the right margin of the sternum; while its lower boundary cannot be delimited by percussion because it is continuous with the dullness of the liver, with which organ the heart is, for all clinical purposes, in close contact.

FIG. 101.



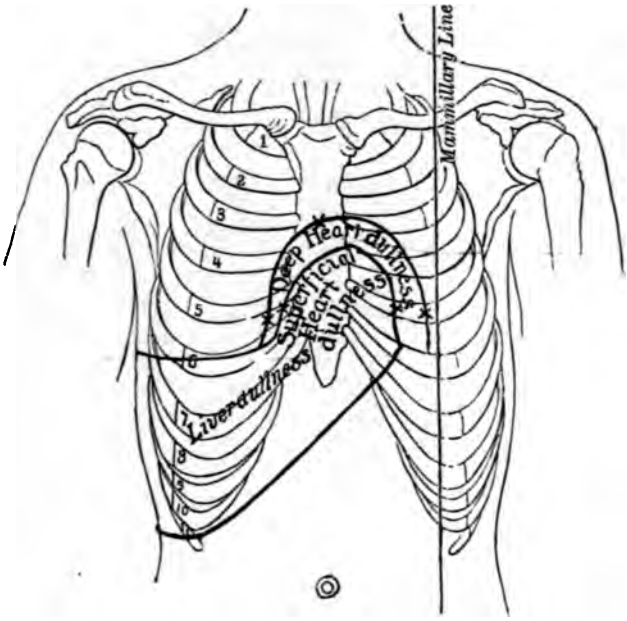
Area of superficial and deep cardiac dullness in normal conditions.—After Sahli.

The area of deep dullness in children does not correspond with that observed in adults, occupying, as it does, a greater relative space. Above, it extends to the second interspace; on the left, to beyond the mammary line; to the right, slightly beyond the sternal border.

Anatomically, the heart projects beyond the right sternal border. No dullness is elicited by reason of this fact. Hence we are pretty safe in asserting that any dullness extending beyond the right border of the sternum is pathological, and may be regarded as evidence of enlargement of the heart in that direction.

Method to be Pursued in Determining Absolute and Relative Dullness.—It is a good rule always, in the practice of percussion, to progress from a resonant to a non-resonant organ. Hence, in delimiting the borders of the heart, one should start from a point situated well within the area of pulmonary resonance. It is also important to remember that when delimiting an organ, the pleximeter (or finger) should be placed with its long axis parallel to the particular edge of the organ, the position of which is to be determined. The force used in percussion must be varied according to the object to be attained. In case it is desired to map out the area of superficial cardiac dullness, *i.e.*, the area of the chest against which the heart is in direct contact, the percussion-blows should be light. On the other hand, when the object is the delimitation of the deep cardiac area, the percussion-blows must be comparatively forcible, in order to elicit the dullness beneath the overlapping pulmonary borders.

FIG. 102.

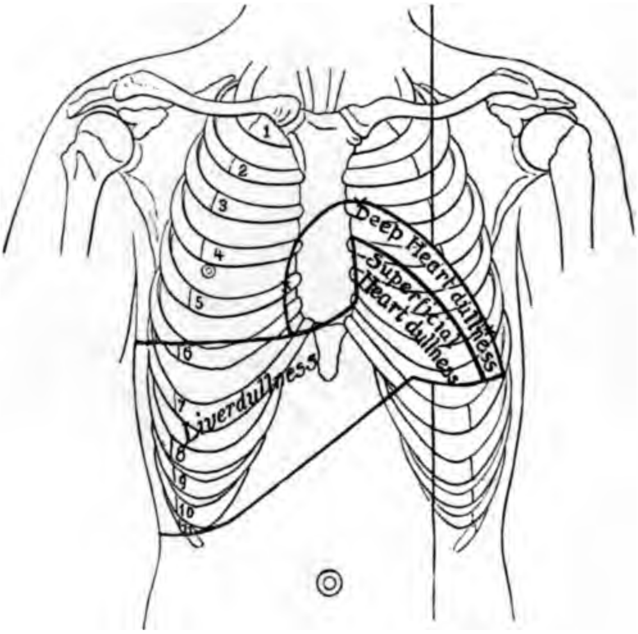


Area of heart-dullness in dilatation of the right ventricle. The increase in area of dullness is almost entirely to the right of sternum.—After Sahli.

To determine the right border of superficial cardiac dullness, we start our percussion over the right lung, the pleximetric finger being placed parallel to the border of the sternum. Care should be taken that we start at a point giving clear resonance. As successive blows are struck, and the character of the sound noted, the pleximeter is brought nearer and nearer to the sternum, until finally a point is reached at which the resulting sound is one of absolute dullness. This marks the right border of superficial cardiac dullness.

To determine the lower border of absolute dullness we must resort to different tactics. We know that the superficial cardiac dullness blends with the adjacent liver dullness. We proceed, therefore, to delimit the upper margin of the liver. Beginning, as before, in the right side of the chest at a point giving pulmonary resonance, but with the long axis of the pleximeter at right angles with the sternum or parallel to the margin of the liver, we percuss and note the condition. Approaching the situation of the liver, we finally reach a line at which dullness appears. Where this line joins the one determined upon as marking the right boundary of superficial cardiac dullness may be regarded as the right extremity of the lower border of the heart. Extending this line to the point of the apex-beat gives us the lower line of cardiac dullness.

FIG. 103.



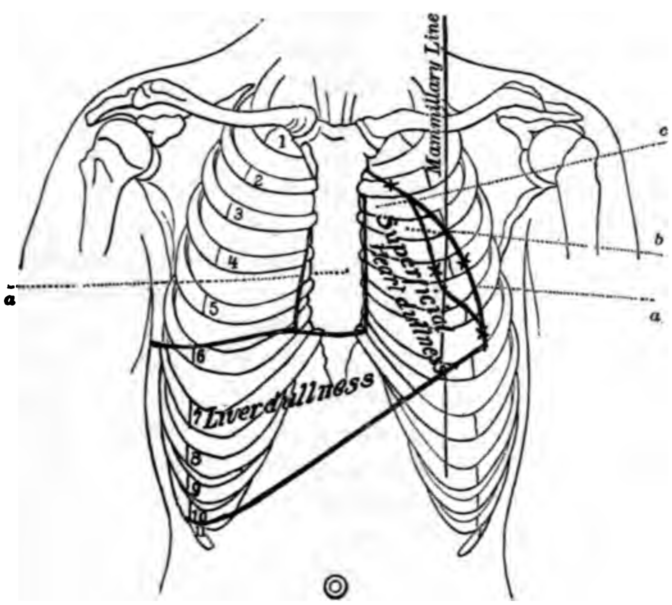
Area of heart-dullness in dilatation of the left ventricle. The increase in dullness is limited to the left side of the sternum.—After Sahli.

To determine the left border of absolute dullness, we proceed in the same manner as we did in determining that of the right. To reach clear pulmonary resonance, we must start our percussion well to the left of the mammary line. It is important to bear in mind that, starting our percussion at the level of the third or fourth rib, we are not obliged to commence as far to the left to obtain pulmonary resonance as we do on the level of the fifth interspace. It will be noted that the normal line of absolute cardiac dullness corresponds closely with a line, with its convexity outwards, drawn from the mid-sternum at the level of the fourth costal cartilage to the apex-beat.

To determine the area of deep cardiac dullness, proceed as above when delimiting the area of superficial dullness, but make the percussion-blows more forcible, in order to overcome the resonance of the overlapping lungs.

The delimitation of the upper border of the heart is not made as readily as might be imagined at first sight, because of the presence in this locality of the great blood-vessels, and of the alterations in the percussion-sounds by the sounding-board property of the sternum. By firm percussion one can define that portion of the upper border of the heart which lies to the left of the great blood-vessels. Laache claims that the upper portion of the heart can be delimited by percussing lightly from above downwards over the left half of the sternum.

FIG. 104.



Area of heart-dullness in dilatation of left auricle and left ventricle, with uncovering of the pulmonary artery. (a) Deep cardiac dullness, right and left ventricles; (b) superficial dullness, the left auricle; (e) dullness due to pulmonary artery.—After Sahli.

When determining the left border of the heart, it is important always to mark out the shape of the apex, *i.e.*, whether pointed or rounded.

The area of cardiac dullness may be either increased or diminished, or it may be altered in its position.

large area of dullness, which approaches a square in contour. The area of dullness may extend from the first or second costal cartilage above to the sixth rib or interspace below. It will furthermore be found that, where the area of relative dullness does not exist, the transition from pulmonary resonance to dullness is sudden. Position of the patient exerts a modifying influence on the percussion signs. When recumbent, the lungs return somewhat to their normal position ; hence there is an increase of the area of pulmonary resonance. The effusion necessarily varies in quantity from day to day, and hence the results of percussion will present daily variations.

(*c*) **Retraction of the Overlying Pulmonary Tissue Because of Fibroid Changes or Pleuritic Adhesions.**—The general character of the percussion changes in this case will be found to be an increase in the superficial or absolute dullness with decrease of relative dullness, the outer margins of the latter, however, remaining in the normal position. In other words, the heart is normal as to size and outline, but a larger portion of the organ than normal is in direct contact with the thoracic walls. Essential to a correct diagnosis under these circumstances is the presence of symptoms demonstrating the existence of disease of the respiratory apparatus, the physical signs of pulmonary changes, and a history of pleurisy, pneumonia, etc.

(*d*) **Extension of the Cardiac Dullness Because of Solid Growths or Structures in Contact with that Organ.**—Under these circumstances, the dullness due to the heart substance is the same in character and extent as in health. But the presence of tumors in comparatively close contact with the heart, or tissue-changes in adjacent organs, *i.e.*, the lungs, as in pneumonia or pleurisy with effusion, brings about an apparent increase of the cardiac dullness, because the dullness due to these pathological conditions is continuous with the normal cardiac dullness. Here, again, we are dependent upon the history of the case, the presence of pulmonary symptoms, and the associated physical signs, for an accurate diagnosis.

Diminution of the area of cardiac dullness may be due to

a. Decrease in the size of the heart (atrophy).
b. Encroachment of lungs on the superficial cardiac space.

(*a*) **Decrease in the Size of the Heart.**—Under this heading may be included those cases of congenitally small heart, and the far more common instances in which the heart atrophies as the result of constitutional wasting diseases, as diabetes, tuberculosis, cancer, insanity, Addison's disease, and amyloid degeneration of the kidneys. This cardiac atrophy is the rule rather than the exception in cancer, diabetes, and Addison's disease. The physical signs in all these cases consist of diminution of the areas of cardiac dullness, together with the usual evidences of weakened heart-action. Necessarily there must also be associated all the evidences of diminished blood-supply to various viscera. The clinical recognition of atrophied heart has an important bearing on prognosis, as the diseases in which it occurs, notably pulmonary tuberculosis, never follow as favorable a course as they do when the heart is acting with its normal energy.

(*b*) **Encroachment of the Lungs upon the Superficial Cardiac Area.**—Under these circumstances, deep percussion demonstrates that the heart is normal in size; while light percussion shows that the superficial area of dullness is diminished. Emphysema of the lungs is the most commonly-observed cause of this state of affairs. The diagnosis here rests upon the characteristic shape of the chest, the expiratory dyspnœa, the hyper-resonance on pulmonary percussion, etc.

When the lungs are made to encroach upon the cardiac area by reason of pressure exerted by various tumors, then the diagnosis is based entirely upon the physical signs, which must, per force of circumstances, vary with the peculiarities of the case under observation.

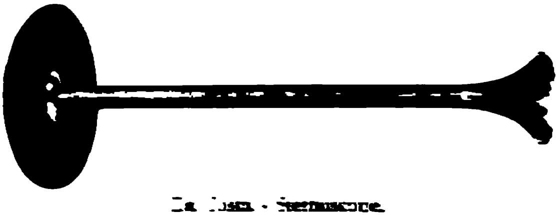
Alteration in the Position of Cardiac Dullness (*Displacement of the Heart*).—Of course, a displacement of the heart necessitates a corresponding alteration in the position of cardiac dullness. Percussion, however, is not of itself capable of discovering this state of affairs, because the condition which produces the displacement is such as to give rise to a percussion-dullness which encroaches on the cardiac area. One is obliged, therefore, to bring to his aid other diagnostic helps. The conditions producing displacement of the heart are: Aneurysmal and other thoracic tumors, pleuritic effusions, pneumo- and pyothorax, retraction of a lung, ascites, abdominal tumors, dilatation of the stomach, etc.

4. **Auscultation.**—Auscultation of the heart may be immediate or mediate; that is to say, we study its sounds by listening with the ear applied directly to the chest over the cardiac region (*immediate auscultation*), or we may listen through an instrument known as the stethoscope (*mediate auscultation*). The former is useful in enabling us to determine the condition of the heart-sounds as a whole; the latter is essential for the correct appreciation of individual elements of the cardiac sounds.

As to the form of stethoscope which is the most generally useful, opinions will always differ. Most of the instruments in general use are good. Each man will decide, however, in favor of the form to which long usage has accustomed him. Of late years the use of the single stethoscope has died out in great measure. This is unfortunate, for it possesses one advantage not afforded by any other instrument, namely, that of enabling the sense of touch to perceive the cardiac impulse while the ear is listening to the sounds. The deservedly popular forms of this instrument include the old-fashioned wooden stethoscope and the metal tube, with hard rubber ear-piece, suggested by Da Costa.

Of the double instruments, also known as binaural stethoscopes, varieties without number have been candidates for professional favor. Personally, I much prefer Paul's pneumatic stethoscope as modified by Dr. Rand. (*Vide* Fig. 106.) Its advantages are lightness of construction; its fixation to the chest by pneumatic pressure prevents the production of friction-sounds between its bell and the wall of the chest, and the strict localization of sounds because of the surrounding chamber containing rarefied air. Unfortunately, it cannot be used on emaciated or hairy chests.

Fig. 105.

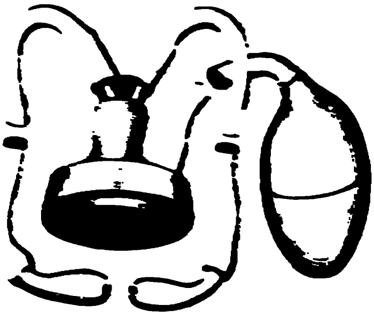


Da Costa's Stethoscope.

The *phonendoscope* is also a very useful instrument. It can be used with advantage over the clothing, though it is far better to apply it next to the bare skin. It can be used with less disturbance to the patient than any other instrument, owing to the flatness of the chest-piece and the great flexibility of the tubes. It gives a most excellent general idea of the condition of the parts beneath, but is deficient in its powers of localization of sounds.

Whatever instrument is selected should have ear-pieces well-adapted to its owner's needs. It may well be said that one should select an instrument to fit with the same care that he would purchase a comfortable pair of shoes.

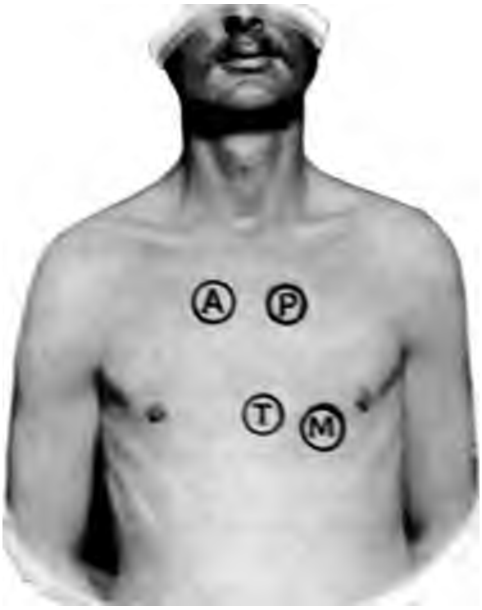
Fig. 106.



Paul's Pneumatic Stethoscope.

Auscultation demands a thorough knowledge on the part of the physician of the various sounds of the normal heart. With this foundation only can he understand the alterations effected by disease. To the ear, the normal sounds of the heart are two in number, viz.: a long or first sound, to which we are listening. It is wise, however, not to depend too much upon such impressions, and, when in doubt in the slighest degree, to be sure that we are right by making simultaneous use of the senses of touch and hearing by feeling the apex-beat while practising auscultation. The uninitiated, at least, should do this invariably. This advice is especially important when investigating the character of cardiac murmurs. Some authorities recommend that the systolic sound be determined by feeling the radial or the carotid pulse. Such advice is bad so far as the radial pulse is concerned, for its impulse is not simultaneous with the apex-beat. The carotid pulse is sufficiently so for all practical purposes, when there is good reason for not observing the time of the apex-beat.

FIG. 107.

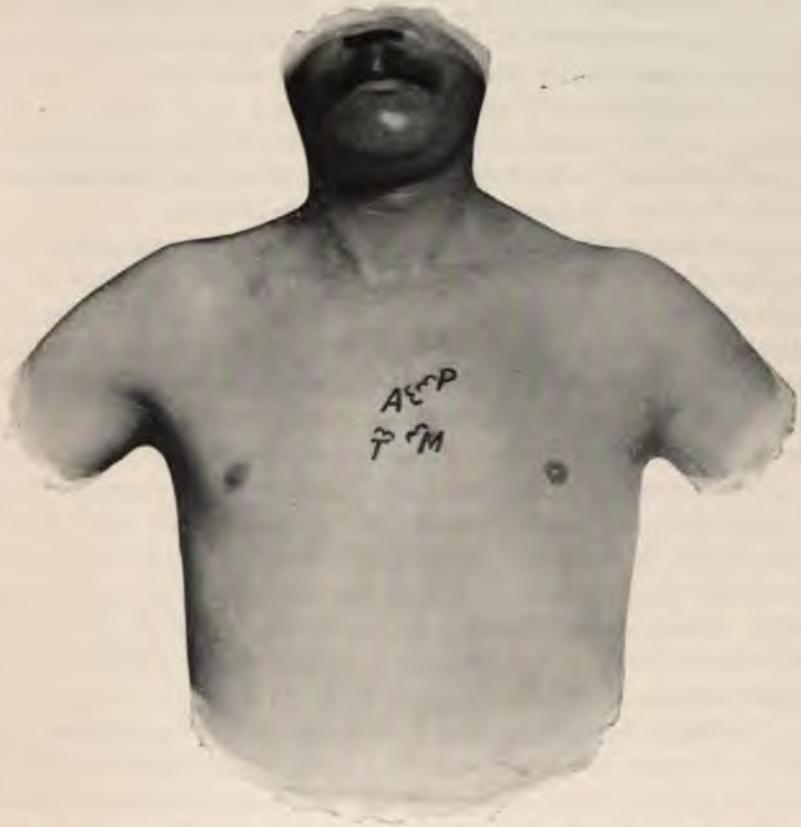


Showing the areas over which the sounds of the respective valves are best heard. A, aortic area; P, pulmonary area; T, tricuspid area; M, mitral area.

The character of the first sound of the heart, as heard at the apex, has already been described as "*booming*."

That of the *second sound* is best described as "*snapping*," being produced by tension of the aortic and pulmonary valves. If one auscultates over the pulmonary and aortic areas, he can separate this sound into two elements, derived from each of these valves. He notices, moreover, that the first sound is not perceived as clearly as it was over the mitral area or apex of the heart. As to the relative intensity of the individual elements of the second sound, authorities differ. One asserts that the second sound is the louder; others contend for the opposite. Careful observation teaches that no absolute rule covering this point can be formulated, beyond the fact that the pulmonary second sound is more prominent than the aortic in early life; while advancing years brings with it comparative accentuation of the aortic element.

FIG. 108.



Showing anatomical relations of valves to surface of chest. A, aortic valve; P, pulmonary valve; T, tricuspid valve; M, mitral valve.

Alterations in Intensity of the Normal Cardiac Sounds.—The sounds of the heart may be increased or diminished in intensity. Such alterations may relate to the cardiac sounds as a whole, or to one or more of them.

Increased Intensity of All the Cardiac Sounds.—This may result from

a. Thinness of the chest-walls.
b. Conditions of the lungs favoring the conduction of sound, *e.g.*, pulmonary consolidation, pneumothorax, etc.

c. Pericardial adhesions.
d. Hypertrophy of the heart.
e. Over-action of the heart from nervous causes.

Diminished Intensity of All the Cardiac Sounds.—This may result from

a. General exhaustion.
b. Thickness of the chest-walls. In women, large mammary glands greatly weaken the intensity of the heart's sounds.
c. Emphysema of the lung, by interposing lung substance between the heart and the wall of the chest.
d. Fluid or air in the pericardial sac.
e. Degenerative changes of the heart-muscle, as in myocarditis.

Alterations in the First or Systolic Sound of the Heart.—These include alterations in quality and alterations in intensity.

First as to **alterations in quality.** The first sound of the heart has been described as "booming." It is made up of two qualities—one of tension, and the other of prolongation,—both of which require attention in clinical work. Prolongation and dullness of the first sound are indicative of hypertrophy of the left ventricle. It is a mistaken idea to assume that cardiac hypertrophy is associated with increased strength of this sound. *It is "prolongation" that constitutes the important clinical sign.*

Dilatation of the left ventricle, on the other hand, causes the *first sound to become short and evince the quality of tension.* At the same time it becomes louder than normal. Indeed, the shortness of the first sound and the increase of the element of tension may cause the first sound to approximate so closely to the characters of the second as to make it indistinguishable from the latter, unless its proper position in the cardiac cycle is determined by the finger over the apex-beat.

A **sharp, slapping, first sound** is observed associated with the presystolic murmur of mitral stenosis. This peculiar alteration of the sound is due to the contraction of the ventricle on an imperfectly-filled cavity.

Increase of intensity of the systolic sound is observed in any condition productive of increased cardiac action, as exertion, excitement, and the early stage of febrile disorders.

Feebleness of the first sound is often incorrectly interpreted as indicating weakness of the heart. In order that it should have this indication, it is necessary that shortness in duration be added to the element of weakness. Such a state of affairs may be observed in the late stages of fevers, especially of typhoid fever, when the so-called granular degeneration of the heart-muscle takes place. Under these circumstances the muscular element may be so completely lost as to leave only the short sound of tension of the mitral valves discernible. The same state of affairs occurs in chronic myocarditis.

Weakness of the mitral first sound, without any change in its duration or pitch, may be due to fatty heart, emphysema, or pericardial effusion.

Increased Intensity of the Aortic Second Sound.—The aortic second sound, depending upon the tension produced by the closure of the aortic valves, must be accentuated in any and all conditions which produce high arterial tension. Hence we note this change in chronic interstitial nephritis, arterio-sclerosis, hypertrophy of the heart, and aneurysm or dilatation of the arch of the aorta.

Agencies which increase the intravascular tension temporarily also cause accentuation of the aortic second sound. Thus, this sign may be observed when the arterioles and capillaries are contracted in rigors, epilepsy, and other convulsive disorders ; sometimes in hysteria, and in the toxæmia of certain of the acute infectious diseases, as scarlatina.

Weakness of the Aortic Second Sound.—Weakness of the aortic second sound is brought about by conditions the reverse of those obtaining when that sound is accentuated. Hence we find it present when blood-pressure is lower than normal. Circumstances bringing this about include weakness of the walls of the left ventricle, relaxation of the arterio-capillary system, as in fevers, especially typhoid, and mitral regurgitation. It may also be observed in connection with strong ventricular contractions. It is then indicative of swelling or thickening of the valvular segments, which do not close with their normal sharpness, though not permitting regurgitation. It is always to be regarded as an unfavorable sign, as it is apt to occur in association with obstruction of the coronary circulation. Inquiry will discover a history of breathlessness on exertion, præcordial distress, etc. Musser suggests that this sign is indicative of atheroma.

Increased Intensity of the Pulmonic Second Sound.—Accentuation of the pulmonic second sound results from any cause which increases the pressure in the pulmonary artery. Hence it is frequently encountered in all conditions which induce pulmonary congestion, including acute inflammatory diseases of the lungs, emphysema, chronic bronchitis, and phthisis. Attention to this sound in the course of pneumonic fever is of the greatest importance, as by it alone can we determine the state of the pulmonary circulation. In the early stage of croupous pneumonia the pulmonic sound is sharp and clear, showing that the right heart is able to overcome the increased intravascular pressure. The loss of this accentuation in the course of the disease will have different significances, according to the associated symptoms. If, at the same time, respiratory distress diminishes, decrease of pulmonary obstruction is indicated. If, on the other hand, dyspnœa increases, we know that the right ventricle is becoming exhausted as shown by its labored beat and the visible pulsations of the right auricle.

Accentuation of the pulmonic second sound occurs also with mitral disease, whether obstructive or regurgitant. Too much importance must not be attached to such accentuation as evidence of mitral disease, unless distinctly confirmed by other signs and symptoms. Especially does this remark apply to the diagnosis of mitral diseases in the young, for in them there is nearly always accentuation of the pulmonic second sound as compared with the aortic. Accentuation of the pulmonic second sound in the course of valvular disease is to be regarded as a favorable sign.

Weakness of the Pulmonic Second Sound.—Weakness of the pulmonic second sound is of importance, and offers an unfavorable prognosis in two conditions. Occurring after accentuation, it shows that the right heart is undergoing dilatation with tricuspid regurgitation ; occurring in pneumonic fever, with coexistent increase in respiratory distress, it also shows failure of the right heart.

Abnormal Sounds.

Thus far our study of the information to be derived by auscultation of the heart has related to the normal sounds and their modifications. It is next in order to study the abnormal sounds. These may be enumerated as follows :

1. Pericardial friction-sounds.
2. The so-called murmurs, endocardial in origin.
3. Hæmic murmurs.

1. **Pericardial Friction-Sounds.**—The characteristic features of the pericardial friction-sound are as follows : Inasmuch as it results from the rubbing of roughened pericardial surfaces against each other by the cardiac movements, it is both diastolic and systolic in time, and hence has been described as "*a to and fro*" sound. Exceptional cases occur in which this feature is not observed. For example, it may present remarkable changes in its rhythm, being "to and fro" one time, and systolic at another. Then, too, we may observe cases in which a triple rhythm is plainly heard. This phenomena is due to friction of opposing pericardial surfaces during (*a*) auricular systole, (*b*) ventricular systole, and (*c*) ventricular diastole. Still more exceptional is a quadruple friction-sound, friction during auricular diastole furnishing the additional sound.

The pericardial friction-sound has been compared to the noise produced by the creaking of new leather. It may, however, be of a softer quality, and then may be compared to the sound produced by gently rubbing together the palms of the hands.

Its intensity is readily altered by a change in the posture of the patient. Sometimes it may be audible only when the patient assumes some particular position. The special attitude favoring its audibility is by no means the same in all cases. In one patient it is most readily discernible when lying down ; in another when bending forwards ; in still another, when reclining backwards, etc. Attempts have been made to show that special qualities of sound indicate specific conditions of the pericardium; and, while a study of this phase of the subject may be productive of practical results, it seems to me that the same end may be attained with greater certainty by the associated physical signs and rational symptoms.

The pericardial friction-sound gives most distinctly an idea of its superficial origin. This sign is especially valuable as differentiating it from endocardial murmurs.

It is audible only over the space occupied by the heart. It has no lines of favorite transmission, as in case of certain valvular murmurs. It may be heard for a short distance below the heart, owing to its conduction by the liver. Its point of greatest distinctness is generally in the third and fourth interspaces.

It disappears with the advent of pericardial effusion, which latter condition is readily discovered by percussion. It returns when the effusion has been absorbed. It usually appears in the earliest stages of pericarditis. With recovery, it disappears gradually, by reason either of formation of adhesions, the complete absorption of exudate, or the disappearance of pericardial roughness by repeated friction. Sometimes the pericardial friction remains permanently, in such cases indicating patches of thickening.

Pericardial friction-sounds may be differentiated from sounds of similar character originating in the pleura by the fact that they continue when the patient ceases his respiratory movements; moreover, it is cardiac and not respiratory in rhythm. Its differentiation from a sound produced by the rubbing of the pericardium against an adjacent inflamed pleura is not so easy. In this case we depend for a correct appreciation of the state of affairs upon the location of the sound (which in *pleuro-pericardial friction* is over the borders of the ventricles, especially the left), and the effect of respiratory movements, cessation of which, by directing the patient to hold his breath, causes the murmur to disappear, while a deep inspiration intensifies the sounds.

Pulsating Vesicular Murmurs.—These occasionally give rise to a mistaken diagnosis of valvular disease. They are produced by the impact of the heart against an overlying margin of lung driving out the contained air. Such sounds are not heard with every cardiac cycle, but only with such as happen to take place at the particular moment at which the overlapping lung is distended with air.

When the portion of lung acted upon in this way happens to be the seat of disease, peculiar râles may be produced with each impact of the heart.

Water-Wheel Sounds.—These consist of peculiar churning sounds, produced evidently by the action of the heart. They indicate the presence of both air and fluid in the pericardial sac. The pathological conditions in which it may occur include perforating wounds of the pericardium, and perforation of the pericardium in the course of œsophageal ulcer, malignant disease of the mediastinum, and septic pericarditis.

Conditions Associated with Pericarditis.—In the majority of cases pericarditis is dependent upon acute inflammatory rheumatism, it being noted as a complication in about one-sixth of the cases of that disease. While usually characterized by severe pain, it may occur without a single subjective symptom to suggest the necessity of examining the state of the heart. This last remark applies especially to the pericarditis of children. Pericarditis also occurs in the course of chronic renal disease, and is then to be regarded as an exceedingly dangerous complication. Tubercular pericarditis may occur, but only in conjunction with evidences of tuberculosis elsewhere.

2. **Cardiac Murmurs.**—It is difficult—indeed, well-nigh impossible—to present in written symbols the peculiar sounds heard and recognized as valvular murmurs. The normal valvular sounds have already been described as sounds giving the idea of tension. The murmurs give the idea of vibration.

The Nature and Method of Production of Murmurs.—Blood flowing through a vessel of even calibre and smooth walls gives rise to no sounds. If, however, that vessel is narrowed in any portion of its course, the impeded current sets up vibrations in the vascular walls, and these may be perceived by auscultation with the stethoscope. Again, if the blood when flowing through a vessel enters a dilated portion, *e.g.*, an aneurysm, the eddies thus set up produce vibrations, and these are heard as murmurs. Bearing in mind the above facts, it is readily conceived that murmurs may be produced at the cardiac orifices (*a*) when they, or the valves which guard them, are roughened; (*b*) when the valves fail to close at the proper time, and permit a regurgitation of blood; (*c*) when the orifices are narrowed; and (*d*) when the orifices themselves are dilated, thus producing a virtual valvular incompetency.

Murmurs are characterized as *regurgitant* when produced by inability of the valves to close at the proper time; and

Stenotic, when due to orificial obstruction.

In order to determine the origin of a given murmur, it is important that the observer determine the following points concerning it:

a. Its point of maximum intensity.
b. Its place in the cardiac cycle.
c. Whether transmitted or not; and, if transmitted, the direction in which it is heard.
d. The way in which it is influenced by exertion, respiration and position.

(*a*) **The Point of Maximum Intensity of the Murmur.**—Murmurs originating at any one of the cardiac orifices are heard with most distinctness in the particular area already designated as the one in which the normal sounds proceeding thence are heard. Thus mitral murmurs are best heard at the apex, aortic at the aortic area, etc.

(*b*) **The Position of the Murmur in the Cardiac Cycle.**—By determining this point, one is enabled to say whether a given murmur is due to regurgitation or stenosis. If one should depend upon auscultation alone for an opinion, he may fail to reach a correct conclusion. He should, therefore, make it a point to feel always for the apex-beat, and note its time as compared with that of the murmur under investigation. If the murmur takes place at the same time as the apex-beat, it is a systolic murmur; if otherwise, it is diastolic. If we determine that the murmur is systolic in time, and is heard with maximum intensity over the mitral area, we conclude that it is due to *mitral regurgitation.*

If it is systolic in time, and heard over the pulmonary area, it is due to *pulmonary stenosis.*

If it is systolic in time, and heard best over the aortic area, it indicates *aortic stenosis.*

If it is systolic in time, and heard over the tricuspid area, it indicates *tricuspid regurgitation.*

If it is diastolic in time, and is heard best over the mitral area, it indicates *mitral stenosis.*

If diastolic in time, and is heard over the aortic area, *aortic regurgitation.*

If diastolic in time, and heard best over the pulmonary area, *pulmonary regurgitation* is suggested.

If diastolic in time, and heard best over the tricuspid area, it indicates *tricuspid stenosis.*

(*c*) **Direction of Transmission of the Murmur.**—Murmurs are transmitted in accordance with the principles governing the conduction of sounds either in the direction of the movement of the column of blood or by the solid tissues set in vibration by such column. The direction of transmission can only be determined by using a stethoscope with a small bell, so as to insure correct localization.

Mitral Murmurs.—*Mitral systolic or regurgitant murmurs* are transmitted from the apex to the left into the axilla, and may be heard even at the angle of the scapula. *Mitral presystolic* or *stenotic murmurs* are usually not transmitted. Their area of maximum intensity is quite limited—almost always to the apex or a little to the inside of the apex. They may, however, be transmitted to the axilla and angle of the left scapula, but such transmission is to be regarded as exceptional.

Aortic Murmurs.—The *aortic systolic* or *murmur of aortic stenosis* is transmitted upward along the line of the vessels, this being the direction of the blood-current. The *aortic diastolic* or *murmur of aortic regurgitation* is transmitted by the walls of the left ventricle down along the line of the sternum even to the apex.

Tricuspid Murmurs.—Tricuspid murmurs are not transmitted. That due to stenosis is presystolic in time, and is usually associated with a similar murmur originating at the mitral orifice. Owing to this frequent association, tricuspid stenosis is not readily recognized. For a positive diagnosis of the condition one must rely upon other symptoms than those furnished by auscultation, prominent among which stands distention of the jugular veins with little or no pulsation. The murmur of tricuspid incompetence may be heard outwards towards the apex, and indeed may extend to that point, in which case it is liable to be mistaken for a mitral murmur. To be indicative of tricuspid insufficiency, such a murmur (*tricuspid systolic*) must be constant, and not of occasional presence. The association of venous pulsation constitutes an important confirmatory sign of tricuspid regurgitation.

Pulmonary murmurs may be safely stated to be not transmitted. That of pulmonic stenosis is conducted along the branches of the pulmonary area, and is audible over a large area of the chest, both front and back.

(*d*) **The Influence of Exertion, etc., upon Cardiac Murmurs.**—The observer is frequently impressed with the fact that cardiac murmurs which are audible at one time are not to be heard at another. The influences which bring about such a state of affairs include alterations in position, exertion, the effect of pressure of the stethoscope, and changes in the efficiency with which the cardiac muscle does its work. Ordinarily, we are inclined to attribute the disappearance of a murmur to improvement in the patient's condition, and generally with good reason ; because exudates may be absorbed, or compensation of the valvular anomaly may be completed. In certain slight lesions, the murmur may be inaudible until the patient gets up and walks around the room a few times ; others, generally of a severe type, become more prominent when the patient is at rest.

A certain amount of strength on the part of the ventricular contractions is required to elicit a murmur ; hence it happens that in cases in which the cardiac muscle is becoming weaker day by day, the murmur disappears accordingly, simply because there is not sufficient muscular contraction to generate it.

As to the influence of stethoscopic pressure upon the murmur, Musser has tabulated the following :

(*a*) **Firm pressure removes**

1. Hæmic murmurs over the base of the heart (save Jenner's pulmonary murmurs).
2. An aortic obstructive murmur of the apex.

3. When mitral and aortic regurgitant murmurs coexist, the aortic murmur is diminished in the greater degree.
4. Aortic regurgitant murmurs over the second right intercostal space.

(*b*) **While it does not markedly affect**

1. Mitral regurgitant murmurs heard over the apex.
2. Mitral obstructive murmurs over the same spot.
3. Tricuspid regurgitant murmurs over the area of greatest intensity.
4. Aortic regurgitant murmurs over the apex.

Mitral Systolic Murmurs.—The mitral systolic is the most frequently observed of all cardiac murmurs. It may arise from either endo- or exocardial causes. In the former case it indicates regurgitation at the mitral orifice. This regurgitation, however, is not necessarily permanent or incurable, as it may be due to what is called a relative insufficiency, *i.e.*, insufficiency dependent upon increase in the size of the mitral orifice, so that the normal valves are no longer competent to effect a complete closure.

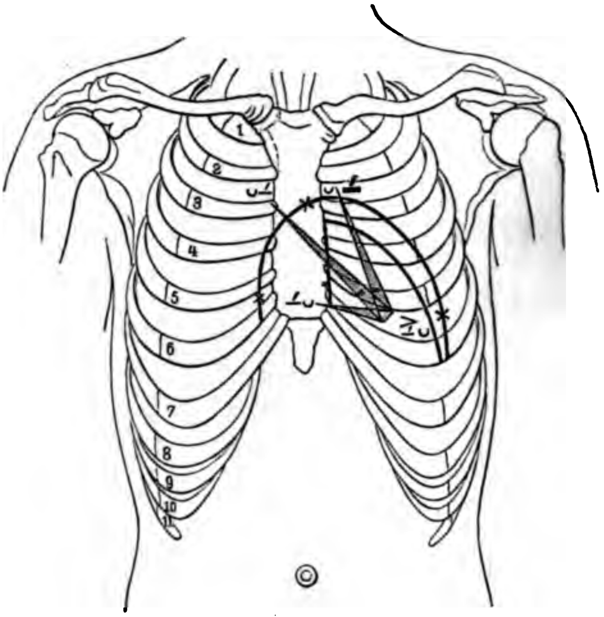
The endocardial causes of a mitral systolic murmur include the following:

a. Damage of the mitral valves by acute simple, ulcerative or malignant, and chronic endocarditis.
b. Stretching of the left auriculo-ventricular orifice from dilatation of the left ventricle, which, in turn, may be due to
    i. Myocarditis.
    ii. Anæmia.
    iii. Debility.
    iv. Acute febrile disorders.
    v. Muscular strain of the left ventricle.
    vi. Neuro-cardiac disease.

(*a*) **The mitral systolic murmurs dependent upon lesions of the valves** are to be recognized by the location and direction of transmission of the murmurs, and by the history of the case. It is the rule for it to be transmitted to the axilla, or even to the angle of the scapula. The clearness with which it is audible at the latter point is sometimes greater than it is in the mitral area itself.

Walshe has even made the claim that if it is inaudible at the angle of the left scapula, it is seldom the result of structural defect. His position, however, is hardly tenable, as shown by numerous observations to the contrary. Occasionally, the murmur of mitral regurgitation is conducted in other directions than those above indicated. Thus it may be transmitted to the left border of the sternum, a phenomenon which has been explained by Sansom as due to "an exceptional conversion into fibroid or calcareous material of the anterior segment of the mitral valve and the fleshy columns attached thereto." Transmission to the left second and third costal cartilages has been explained by Sansom as resulting from "the existence of an abnormal thickening conducting the vibrations to the interventricular septum close to the origin of the aorta." In case of the presence of either of these irregular directions of transmission, differentiation from the simulating lesion is to be made by the associated signs and symptoms: from tricuspid regurgitation by the absence of venous (jugular) and hepatic pulsation, and from aortic stenosis by the state of the pulse and the transmission of the murmur of the latter along the line of the great blood-vessels.

FIG. 109.



Mitral insufficiency, showing slight hypertrophy of left ventricle, and marked hypertrophy of right side of the heart. Accentuation of pulmonic second sound, indicated by the sign **U ⊥** at the base. Murmur heard at apex with maximum intensity. Transmitted to the left (**>**) and diminishing.—After Sahli.

The character of the murmur itself possesses but limited value from a diagnostic standpoint. It is true that its possession of a musical character, in part, makes its organic origin all the more certain. Its length will vary greatly in individual cases. Sometimes it occupies the entire systole, and in other cases only the last portion of that period. Its character may be utilized, however, in conjuction with other signs, in determining the extent of the incompetence. Other things being equal, limited area of conduction, the preservation of the first sound, and the occurrence of the murmur during the late systolic period, indicate a slight degree of regurgitation, and *vice versa.* It must be remembered that intensity of a murmur depends in great measure upon the strength of ventricular contractions, and the same holds good as to the extent of the area over which it is conducted. Hence, limitation of a mitral systolic murmur to the apex or the parts nearby may arise from this cause. In case of failing compensation it may disappear altogether.

The history of the patient in a case of mitral insufficiency dependent upon endocarditis is of very great value. We find, as a rule, that there has been preceding acute inflammatory rheumatism, scarlatina, measles, or chorea. The importance of rheumatism as a cause of valvular damage is so well recognized that we doubt if any physician ever examines a case of heart disease that he does not make inquiry concerning that disease. It is otherwise with the other affections to which reference has been made above. In the majority of cases of endocarditis complicating acute inflammatory rheumatism, the mitral valves and the portions of the endocardium adjacent thereto are the first involved.

Preceding the actual occurrence of the murmur there is a prolongation of the first sound of the heart, due, in all probability, to an impairment of its valvular element. Should the patient be examined for the first time, when only the cardiac difficulty sends him to the physician, one is often at a loss to determine its cause. In the case of children or infants, one is tolerably safe in attributing the lesion to a rheumatic origin, as it is well known that in them rheumatic endocarditis may run an acute course without special symptoms directing attention to the heart. In the case of young adults, it is also probable that rheumatism is the cause, unless special symptoms indicative of other troubles are present. In persons advanced in the degenerative period of life, we are obliged to assume that the mitral incompetence is sequential to ventricular dilatation, itself secondary to widespread cardio-vascular changes.

In the early stages of mitral regurgitation the murmur as described above constitutes the only physical sign. Later there occurs a series of signs and symptoms which may be readily appreciated when the mechanical effect of regurgitation through the mitral orifice is considered.

The first of these is accentuation of the pulmonic second sound. By this sign is meant not that the pulmonic second sound is louder than the aortic, but that it is accentuated as compared with what we conceive to be the proper standard for that particular individual in health. This pulmonic accentuation depends upon the regurgitation producing increased pressure in the pulmonary circulation. The latter, in its turn, throws extra work upon the right ventricle, which then undergoes hypertrophy; and, when compensation fails, dilatation and general venous stasis follow. The left side of the heart likewise suffers. The regurgitation through the mitral orifice leads to early dilatation of the left auricle, the condition of which is not readily determined by physical examination because of its deep situation. With each contraction this dilated auricle delivers to the ventricle an abnormally large quantity of blood, to take care of which that portion of the heart must also undergo hypertrophy and dilatation. These conditions are readily determined by palpation and percussion.

Compensation of mitral regurgitation may continue for years, the patient apparently being none the worse off for his valvular defect. With ruptured compensation comes venous engorgements leading to congestions, cyanosis, and dropsy. Respiration becomes embarrassed even to the extent of orthopnœa.

Congestion of the liver and spleen is evidenced by great enlargements of these organs. The kidneys likewise take on changes, as exhibited by diminished urinary secretion, albuminuria and casts. Pathologically this renal condition, as well as that of the liver and spleen, is spoken of as cyanotic induration.

The murmur of mitral regurgitation may be mistaken for that of tricuspid regurgitation, functional murmurs, aortic stenosis, and roughening of the aortic valves.

Tricuspid regurgitation is, under the most favorable circumstances, difficult of recognition, as shown by the fact that the vast majority of cases fail of diagnosis during life. Its murmur is heard inside the point of the apex impact, at times, though usually at the base of the ensiform cartilage. As already stated, it is associated with jugular and hepatic pulsations ; it is not transmitted to the axilla or the angle of the left scapula, and is not associated with accentuation of the pulmonic second sound. The real diagnostic difficulties seem to arise in those cases in which tricuspid and mitral systolic murmurs are associated. Then one is obliged to rely upon the differences in pitch of the abnormal sounds as heard over the tricuspid and mitral areas, repectively.

Functional murmurs are not transmitted to the axilla and angle of the left scapula, as are organic mitral murmurs, nor are they associated with the physical signs of the conditions sequential upon organic mitral incompetence.

(*b*) **Mitral Systolic Murmur from Stretching of the Left Auriculo-Ventricular Orifice.**—All of the causes mentioned under this heading (*vide* p. 460) depend for their existence upon the fact that the left ventricle in systole exerts a sphincter-like action upon the mitral orifice. It has been stated that the relative size of the orifice during diastole and systole may be as 13 : 6.7. It is not unreasonable, therefore, to expect mitral incompetence from sheer weakness of the heart-muscle.

The cardiac weakness from muscular degeneration in typhoid fever and other acute febrile disorders is usually manifested by disappearance of the muscular element of the first sound. Only exceptionally is mitral regurgitation suggested by the presence of an apical systolic murmur. In the vast majority of these cases palpation and percussion show no evidence that the heart is dilated in the slightest degree. The area of cardiac dullness is sometimes apparently increased,—owing, doubtless, to emaciation making the underlying conditions more readily obtainable. Sansom makes the statement—which seems hard to accept—that the heart itself is ofttimes smaller than normal.

The *mitral murmur of anæmia and debility* evidently depends upon a variety of factors, including the blood-changes, cardiac dilatation, increased vascular tension, and emotional excitement. It is often present in cases in which the blood-changes are slight or moderate, and absent when the anæmia is profound. Thus it is plain that the quality of the blood alone is not essential to its production. When, however, there is added an exciting cause, *e.g.*, sudden excited action of the heart, as from fright, the orificial changes sufficient to produce mitral incompetence seem to occur. Increased vascular tension by throwing greater strain upon the valves tends in the same direction.

The *mitral regurgitant murmur succeeding severe muscular strain* is due mainly to the dilatation of the left ventricle, the incompetence being relative. In other cases, however, the heart-strain first produces rupture of one of the aortic cusps, thus throwing great labor on the left ventricle, which dilates. Then follows the mitral incompetence. It should be remembered that the majority of cases of heart-strain are not attended by valvular murmurs, the main clinical phenomena consisting of dilatation of the left ventricle, as evidenced by percussion, præcordial distress, and irregular and rapid action of the heart. The murmur, when present, does not exhibit the rough character of the murmur when resulting from organic changes in the valves themselves, but is generally soft.

The clinical phases of cardiac neuroses are as numerous as the individuals suffering therefrom. In the vast majority no objective symptoms other than arrhythmia and tachycardia are manifest. Occasionally, however, dilatation ensues, this being especially true of exophthalmic goitre. This of itself is capable of producing relative mitral incompetence. The defective innervation of the heart naturally brings about weakness of muscular action, and this, in its turn, lessens the sphincter-like function of the mitral orifice, thus again producing regurgitation at that point. The histories of patients with cardiac neuroses and the absence of evidence pointing to an organic cause make their recognition an easy matter.

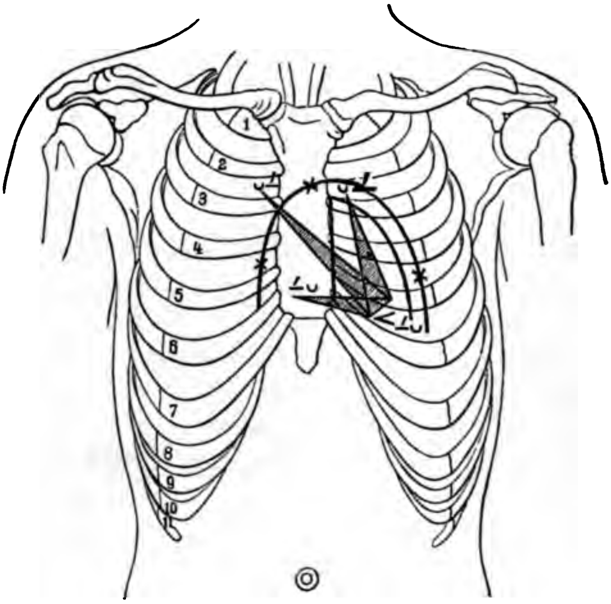
Mitral Presystolic Murmur.—A presystolic murmur audible in the mitral area may occur in

a. Mitral stenosis.
b. After an attack of pericarditis in children.
c. Occasionally in aortic regurgitation.

A presystolic murmur due to tricuspid stenosis may have its point of maximum intensity so near the mitral area as to make its diagnosis a difficult matter.

(*a*) **Mitral Stenosis.**—For the diagnosis of this condition one must depend upon the history of the case as well as upon the physical signs. Although the latter are well defined in most instances, they vary so much in different stages of the illness as to make it an easy matter to overlook the valvular difficulty. Errors are made all the more readily because mitral stenosis rarely exists unassociated with other valvular troubles, the physical signs of which are more obtrusive, or at least are subject to fewer variations.

FIG. 110.



Mitral stenosis. Left ventricle of normal size. Hypertrophy of the left auricle and of right side of the heart. Pulmonic second sound accentuated. Maximum intensity within apex: murmur increasing to first sound (<). Not transmitted.—After Sahli.

Mitral stenosis is practically always the result of rheumatic endocarditis. The resulting deformity is of slow formation, and may assume one of two well-known types: (i) The *funnel-shaped orifice*, in which the edges of the valves are united to each other, this variety being specially common in children; and (ii) the so-called *button-hole orifice*, which shows itself as a mere slit where the normal orifice should be. The majority of cases occur in females.

A proper understanding of the clinical features of mitral stenosis demands a knowledge of the mechanical effects of the lesion. In the first place, increased work is thrown upon the left auricle in forcing blood through the narrowed orifice. This cavity therefore dilates, and undergoes more or less hypertrophy. This change does not, however, compensate fully for the difficulty; hence there is obstruction in the pulmonary circulation, to overcome which the right ventricle likewise dilates and hypertrophies, the latter being especially well marked. Indeed, the hypertrophy of the right ventricle is usually so great as to force the left ventricle backwards, so that the apex of the latter is no longer in contact with the thoracic walls. With failure of compensation, the tricuspid orifice becomes incompetent under the strain induced by the right ventricular hypertrophy and dilatation. The left ventricle suffers no changes in pure mitral stenosis other than occasional reduction in size, the result of its imperfect filling with blood through the mitral chink. Inasmuch as mitral stenosis is rarely of a pure type, usually being associated with regurgitation at that orifice, hypertrophy of the left ventricle is commonly observed. *It must be understood, however, that this change is not at all related to the stenosis, but to the regurgitation.* This statement is in direct contradiction to that of Gibson, who claims that the diastole of the left ventricle being an active process, and assisting as it does the onward passage of blood through the left auriculo-ventricular orifice, becomes more energetic than normal in endeavoring to compensate for the obstruction, and hence that part of the heart undergoes hypertrophy. Clinical evidence of others does not bear out the correctness of his theories and observations.

Taking the symptomatology of mitral stenosis as a whole, we find the characteristic features include a presystolic murmur with maximum intensity to the inner side of the apex, but transmitted over a limited area; accentuation of the pulmonic second sound; displacement of the apex-beat to the left, owing to hypertrophy of the right ventricle; and extension of percussion-dullness upwards, even to the second left interspace, owing to hypertrophy of the left auricle.

These symptoms are so distinctive that one would naturally think that the diagnosis of mitral stenosis is a very simple matter. As a matter of fact, such is far removed from the truth, for the auscultatory signs upon which we mainly rely are remarkable for their changeability, being prominent one day and absent the next. Indeed, the frequent association of mitral stenosis with other valvular difficulties leads to failure to recognize it, the complicating condition alone being correctly diagnosed.

If, however, we study mitral stenosis after the manner of Broadbent, dividing its course into three stages, many of our diagnostic difficulties will disappear. *The first of these stages* presents a presystolic murmur heard over a limited area to the inner side of the apex. This murmur is of a rough vibratory character, and may be associated with a plainly-perceptible thrill over the same area. This murmur immediately precedes the first sound of the heart, running up to it, and ending suddenly. The second pulmonic sound is heard plainly at the apex.

The *second stage* begins when the second sound is no longer audible at the apex. That this stage is of practical value is appreciated when the reasons for the disappearance of the aortic second sound at the apex are understood. The hypertrophy of the right ventricle removes the left ventricle from the thoracic walls. The increased stenosis at this period leads to imperfect filling of the left ventricle, and consequently to lessening of the quantity of blood in the aorta. Necessarily, then, the aortic valves close with less energy, and at the same time are heard less clearly because of the increased distance from the examining ear. The murmur itself manifests different characteristics from those observed in the first stage. It becomes longer, so that it occupies a greater portion of the diastolic period. In the first stage the murmur has a gentle onset, increasing gradually up to the moment of systole, when it ceases abruptly. In the second stage it may begin with considerable intensity, gradually decreasing, then increasing through the presystolic period, and ending abruptly with the first sound. Physical signs indicative of enlargement of the right ventricle are plainly manifested by displacement of the apex-beat to the left, and increase of cardiac dullness to the right of the sternum. In this stage, also, the first sound assumes a sharp, sudden, tapping character, the normal sound of prolongation giving place to that of tension. This remarkable change is to be attributed to the imperfect filling of the left ventricle during diastole. When, therefore, the ventricle contracts, it meets with comparatively little resistance until its walls come in contact with the contained blood; thus it meets with sudden tension, and the actual systole is considerably shortened. Hence the sharp and sudden tapping sound.

The *third stage* is characterized by the disappearance of the presystolic murmur, the only auscultatory evidence of the mitral stenosis remaining being the short, snapping sound at the apex above referred to. This stage marks the appearance of advanced changes, the disappearance of the presystolic murmur being contemporaneous with the establishment of tricuspid regurgitation, which prevents the right ventricle from exerting sufficient force to drive the blood through the mitral orifice with sufficient force to generate a murmur.

Mitral stenosis must always be remembered as, next to aortic regurgitation, the most serious of the valvular lesions. It is, moreover, the valvular lesion which, above all others, favors the occurrence of arterial embolism. It may be associated with dropsy, as may other valvular lesions, but, unlike them, the infiltration is inclined to favor the abdominal cavity,—ascites. Mitral stenosis is also apt to be associated with hæmoptysis and congestion of the liver.

(*b*) **Presystolic Murmur in Adherent Pericardium.**—A presystolic murmur, audible at the apex, is occasionally observed succeeding an attack of pericarditis in children. This murmur is usually rumbling in character, and is not associated with accentuation of the first sound at the apex. The history of the case and the transiency of the murmur establish the differential diagnosis. We have also, for guidance, the concurrent signs of adherent pericardium, as follows:

Of importance is the retraction of the thoracic surface with each apex-beat. Sometimes an undulatory movement is observed over the cardiac area. This latter must not be confounded with a similar movement produced by a want of synchronism in the contraction of the ventricles. The characteristic sign, however, is a decided recession of intercostal spaces, ribs and cartilages, with each cardiac systole. Associated with this symptom will be a decided enlargement of the heart, and evidences of conditions capable of producing pericardial adhesions, as pleurisy and pulmonary tuberculosis. It should be remembered that pericardial adhesions, especially in children, make the prognosis much more serious than would otherwise be the case. Even should such patients recover, they are not apt to live to advanced life—rarely after fifty. Nevertheless, it was my fortune to observe one case in which the lesion was unsuspected during life, and yet in whom it must have existed at least from early manhood, and who died suddenly at the age of sixty-seven.

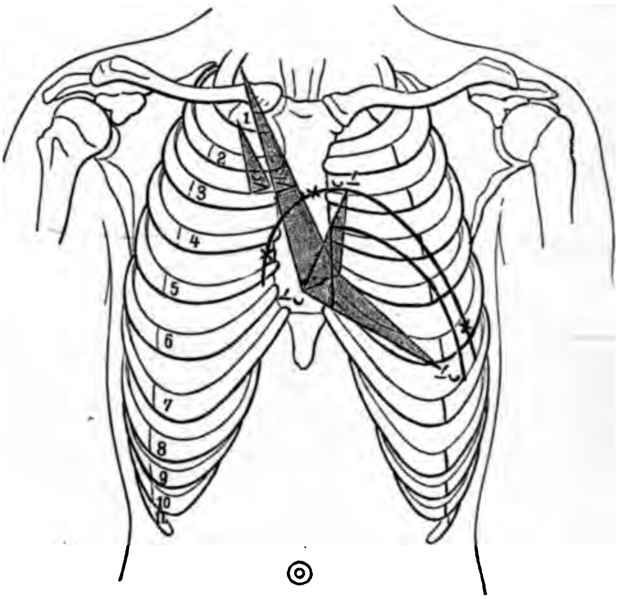
(*c*) **Presystolic Murmur with Aortic Regurgitation.**—The fact that a presystolic murmur, audible in the mitral area, might constitute important evidence of aortic regurgitation, was first noted by Austin Flint; hence the designation of "*the Austin Flint murmur*," as applied to this sign. So far as the characteristics of it and those of the murmur of mitral stenosis itself are concerned, the examiner may be unable to decide. One must rely upon concurrent signs and symptoms. Mitral stenosis is favored by the accentuated pulmonic second sound, the hypertrophy of the left auricle and right ventricle, and the thrill in the mitral area; aortic regurgitation by left ventricular hypertrophy, the water-hammer pulse, etc.

Aortic Diastolic Murmur.—A diastolic murmur audible in the aortic area is indicative of regurgitation at the aortic orifice, and aortic aneurysm. Notwithstanding the clearly-defined indication of this sound, one will make serious errors if he depends upon it solely for the recognition of aortic incompetence, for it is not infrequently absent, while, in many cases, its point of maximum intensity is situated in other than the conventional location. Then, too, there are associated signs and symptoms which, taken together, make the diagnosis absolutely certain, though all murmurs be absent.

The orthodox position for the murmur of aortic incompetence is over the aortic area, *i.e.*, close to the sternum in the right second interspace. From this point it is conducted upwards along the sternal margin to the right sterno-clavicular junction, and downwards along the sternum. It may be audible also at the apex without being discoverable between that point and the points just mentioned. This fact is explained by the intervention of the right ventricle. It is usually soft in character ; as Sansom describes it, it has an aspirating character. It may be either short or long, the latter to the extent of not only replacing the second sound, but filling-in the full period of silence between it and the first sound.

But the aortic area is not always the region in which the aortic regurgitant murmur is the most distinctly audible. Thus it is often heard (*Cabot says in the majority of instances*) on the left side of the sternum, at about the level of the fourth costal cartilage.

FIG. 111.



Aortic insufficiency. Left ventricle greatly hypertrophied. Right side of heart normal in size. Murmur heard over a considerable area, at times with maximum intensity at base in aortic area, at times at lower end of sternum. No accentuation of second sounds.—After Sahli.

Still another point at which it may be heard more distinctly than elsewhere is the left third interspace, exactly over the pulmonary valves or artery.

The fact that aortic regurgitation may give rise to a presystolic murmur in the mitral area has already been mentioned. (*Vide* page 470.)

The associated cardiac changes constitute very important evidence of the aortic insufficiency. No cardiac lesion is capable of producing such remarkable hypertrophy of the left ventricle as is the one under consideration. So great, indeed, is the enlargement, that it has given rise to the name "*cor bovinum.*" The associated hypertrophy and dilatation may be accepted as fairly reliable evidence of the extent of the regurgitation.

The pulse presents the well-known "water-hammer" character. (*Vide* Corrigan's pulse, page 114.) Very commonly present, but not diagnostic, is venous or capillary pulsation. This is especially marked when fever or other conditions producing low vascular tension are present. Indeed, it may be produced by low vascular tension without the aid of this valvular anomaly. When irregularity supervenes, it may be assumed that the case has progressed to an unfavorable stage.

The conditions which may give rise to aortic regurgitation are quite numerous. In the majority of cases it results from atheromatous changes; hence it is commonly observed in subjects of advanced years, especially those who have been addicted to great physical exertion. Very often these aortic changes are syphilitic in origin. It has long been a subject of comment that aortic incompetence occurs with special frequency in association with locomotor ataxia.

Next to atheroma, rheumatic endocarditis is the most frequent cause. When this is the case, the mitral valve will also be found to be diseased.

Traumatism, *i.e.*, rupture from violent physical exertion, affects the aortic more frequently than any other of the cardiac valves. It is not unlikely that in the majority of cases of this accident the valves have been weakened by structural changes originating in the causes above mentioned.

The production of aortic regurgitation by dilatation of the orifice, thus making the normal valves incapable of closing the same, has been asserted, but may well be considered doubtful, for the aortic ring is rigid, and its dilatation by reason of any ordinary circumstances is well-nigh inconceivable.

Aortic regurgitation is to be regarded as offering a very unfavorable prognosis. As long as compensation is maintained, symptoms are absent. With failure of compensation, the usual evidence of cardiac insufficiency, dyspnœa, œdema, etc., appear. In aortic incompetence, pains simulating those of angina pectoris are not uncommon. Indeed, anginoid seizures may appear and result fatally. It is said that aortic regurgitation is the valvular lesion which, above all others, is the cause of sudden death.

The differentiation of aortic diastolic murmur of regurgitation from that due to small aneurysm of the aorta above the valves in the pouches of Valsalva is not by any means an easy matter. Pressure-symptoms are usually absent, because of the small size of the tumor. One must, then, rely upon the history of the case and the associated symptoms. Aneurysm fails to give a history of rheumatism the usual cause of aortic regurgitation, and does tell of syphilitic infection, the commonest cause of aneurysm. The latter lesion is more likely to be attended by cardiac distress, paroxysmal dyspnœa, and præcordial pain. Intra-thoracic aneurysm may also produce a diastolic murmur in other than the aortic area, *e.g.*, near the right sterno-clavicular junction. This murmur is plainly discernible as separated from the heart-sounds. It is due to the elastic recoil of the aneurysmal sac after its distention by ventricular systole; hence it must be diastolic in time. When dependent upon aneurysm of the descending portion of the thoracic aorta, it may be most audible in the interscapular region.

Sometimes this murmur is double, the systolic portion depending upon the inrush of the blood over the roughened edges of the aneurysmal sac.

Systolic Murmur at the Aortic Area.—A systolic murmur audible at the aortic area may be produced by a number of causes, as follows:

(a) Roughening, stiffening, or malformation of the aortic orifice.
(b) Stenosis of the aortic orifice.
(c) Roughening or dilatation of the arch of the aorta.
(d) Pulmonary stenosis.
(e) Open ductus arteriosus.
(f) Mitral regurgitation.
(g) Blood conditions (anæmias).

(*a*) **Aortic Stenosis.**—This is erroneously held to be the most commonly-observed cause of a systolic murmur audible in the aortic area. The latter murmur, to be significant of aortic stenosis, must be attended by certain characteristic phenomena.

The murmur itself is usually most pronounced in the second intercostal space, to the right of the sternum. Nevertheless, its point of maximum intensity may be as high as the right sterno-clavicular articulation or as low as the fifth right interspace. It is a long murmur, continuing to the end of the ventricular systole. Its area of transmission is extensive, especially along the line of the great blood-vessels. Hence it is audible in the axillary and carotid arteries.

As a rule it is harsh and loud, but it may be musical. Its character and intensity will vary from time to time, according to the strength of the ventricular systole. When the latter weakens, the murmur becomes weaker, and it may be softer.

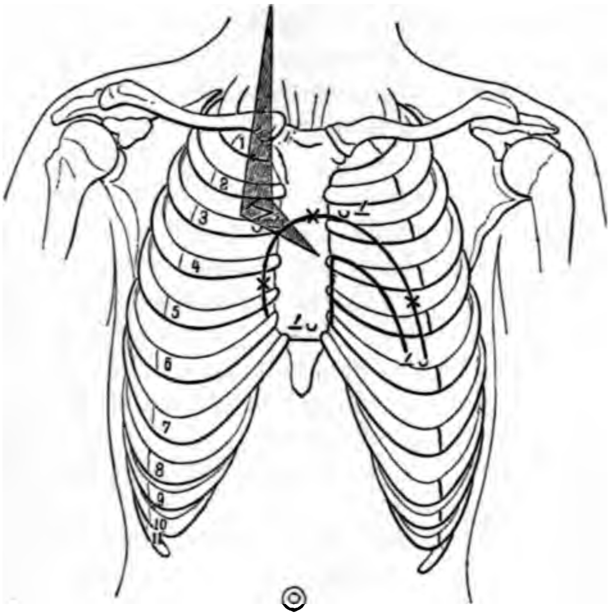
It is a serious mistake, however, to depend upon this murmur for our diagnosis of aortic obstruction. Neglect of this is the main cause of the frequency with which aortic stenosis has been diagnosed, and has led to the prevalent view that this valvular anomaly is of comparatively favorable prognosis. To make the diagnosis of aortic stenosis certain, the following symptoms must be associated with a systolic aortic murmur:

(*i*) *The pulse presents certain characteristics*, as follows: Owing to the obstruction the ventricle empties itself slowly, and this produces a correspondingly slow rise in the pulse-wave. The acme of the pulse-beat is well-sustained, hence sphygmographic tracings exhibit a broad summit.

The gradual entrance of blood into the arteries necessarily causes this wave to be of less than the normal height. The descending stroke, like the ascending wave, is gradual. The pulse is also infrequent, owing to the delay in the emptying of the left ventricle. Sometimes the pulse-tracing exhibits the characteristics which have elsewhere been described as the anacrotic curve.

Characteristic as are these pulse-changes, they must not be accepted as pathognomonic of aortic stenosis, for the slow, long pulse, with flat summit, is sometimes seen in mitral stenosis, and is not infrequent in renal disease, but, taken in association with the other symptoms, is invaluable in determining obstruction at the aortic orifice.

FIG. 112.



Aortic stenosis. Left ventricle somewhat hypertrophied. No changes on right side of heart. Murmur heard with maximum intensity in aortic area, transmitted up into cervical vessels.—After Sahli.

(*ii*) *Weakness of the aortic second sound* is the rule when an aortic systolic murmur is due to stenosis at that orifice. Such weakening is readily explained. The gradual entrance of the blood into the aorta leads to a less energetic recoil with the commencement of ventricular diastole; hence the aortic valves close more gradually, and so give rise to a less intense second sound.

(*iii*) A *thrill* is often felt over the upper end of the sternum, especially over the second right interspace. It is systolic in time, it may be transmitted to the carotids, and is sometimes diffused over a wide area of the chest. This symptom likewise is not pathognomonic, as it may be observed in some cases of aortic aneurysm, but emphasizes the diagnosis of aortic stenosis when suggested by the characteristic murmur and the pulse already described.

(*iv*) *Heart Changes.* Aortic stenosis, by throwing increased labor upon the left ventricle, necessarily brings about hypertrophy of that structure. Such enlargement never proceeds to the high degree already described as attending aortic regurgitation. The apex-beat is usually displaced downwards by about one interspace, sometimes more, but is rarely displaced outwards. The ventricular impulse is well-defined, and has been described by Broadbent as a "deliberate push of no great violence." Such systolic action is associated with a first sound which is dull and prolonged. The etiological factor must also be accepted as of diagnostic value. The majority of cases of aortic stenosis result from atheroma, hence it is more commonly observed in advanced life. The most marked cases of stenosis arise from this cause. The pathological process is of such slow progress that it may continue for years without exciting symptoms. Sometimes the vascular changes producing aortic stenosis originate in gout, syphilis, alcoholism, chronic renal disease, or muscular exertion, in which case the changes proceed more rapidly than when senile degeneration is the cause, and the symptoms are correspondingly more obtrusive. Acute endocarditis may also be a cause of aortic obstruction, but it is very exceptional indeed that the lesion is limited to the aortic orifice, the mitral valves likewise suffering. No matter what the cause, aortic stenosis is exceedingly prone to be associated with aortic regurgitation, the same lesions which narrow the orifice producing deformity of the valvular cusps.

(*b*) **Roughening, Stiffening, or Malformation at the Aortic Orifice.**—Aortic systolic murmur from this cause originates in the vascular degenerations of advancing years. It is the condition which, more frequently than any other, is mistaken for the aortic stenosis. The correct status of the case is readily recognized by the attendant signs of arteriosclerosis, especially by the high arterial tension and the characteristic accentuation of the aortic second sound. The sign last mentioned is especially important for differential diagnosis, as the aortic second sound is practically always weak when obstruction exists at the aortic orifice.

(*c*) **Roughening or Dilatation of the Arch of the Aorta.**—These conditions are associated with an aortic systolic murmur. In the case of dilatation, the differentiation from stenosis is made by the accentuation of the second sound and the presence of an abnormal area of dullness to the right of the sternum.

Roughening of the aorta occurs in persons of advanced years with atheromatous vessels. It is usually associated with a slight degree of dilatation, which adds to the intensity of the murmur. It lacks the thrill and characteristic pulse of aortic stenosis, and there is accentuation of the aortic second sound.

(*d*) **Aneurysm of the Arch of the Aorta.**—The differentiation of this from aortic stenosis may be puzzling, because aneurysm presents all the phenomena of aortic stenosis excepting the weak second sound and the characteristic pulse of that condition. In aneurysm we depend for an opinion upon the presence of a well-marked tactile thrill, pulsation in the second right interspace, and the well-known pressure symptoms, which include pupillary changes, pain, differences in the radial pulses of the two sides, and disturbed function of the recurrent laryngeal nerves. Sometimes the pulsation is observed in the first right intercostal space. The aneurysm is then known to be of the ascending aorta, if the murmur is transmitted into both the carotid and subclavian arteries. When the aneurysm is of the innominate artery, the murmur is audible in the right subclavian and carotid arteries only. Limitation of the murmur to the axilla indicates aneurysm of the subclavian artery.

Of occasional diagnostic value is the transmission of an aneurysmal murmur to the trachea. To detect this, when present, the patient should be directed to take a deep inspiration. The stethoscope is then placed over the manubrium sterni, while the patient expires slowly, with his mouth shut and one nostril closed by finger-pressure. The respiratory sound thus unduly prolonged is broken into a number of short puffs by the aneurysmal pulsations.

Sansom speaks of additional auscultatory evidence of aneurysm, when a murmur over the thorax has been weak or absent, by causing the "patient to place within his mouth the small chest-piece of the binaural stethoscope, and to close his mouth over it. On auscultating thus, the observer may hear a distinct or loud systolic murmur in the case of thoracic aneurysm, the vibrations being communicated to the trachea, and thence directly by the air-column to the ears."

(*e*) **Functional Murmurs, Including Those of Anæmia and Neuro-Cardiac Disease.**—Murmurs originating in anæmia are heard less frequently over the aortic than any other valvular area. When present, they are soft in character and systolic in time. They are not transmitted in any definite direction, hence they are not liable to produce the impression that aortic stenosis is present. It is possible, however, to confound them with the soft murmur produced by slight rheumatic changes of the aortic valves or to papillomata. (Sansom.) The associated symptoms are invaluable in determining the hæmic origin of the murmur, *e.g.*, the age and sex of the patient, and the systematic examination of the blood.

Functional murmurs arising from neuro-cardiac disease, as in exophthalmic goitre, depend for their diagnosis upon a study of the symptomatic totality, *i.e.*, the absence of phenomena belonging to organic aortic disease on the one hand, and the presence of the characteristic symptoms of exophthalmic goitre or hysteria on the other.

(*f*) **Pulmonary Stenosis.**—When the murmur of pulmonary stenosis is in the conventional area, *i.e.*, in the second interspace to the left of the sternum, there is no danger of mistaking it for a systolic aortic murmur. When, as occasionally happens, its seat of maximum intensity is in the aortic area, it is to be differentiated from aortic stenosis by the fact that it is not transmitted in the direction of the great blood-vessels, and that it produces no alteration of the aortic second sound.

(*g*) **Persistent Ductus Arteriosus.**—The murmur of persistent ductus arteriosus starts with the systole, and continues into the diastole. It is associated with a rapid hypertrophy and dilatation of the right ventricle, dilatation of the pulmonary artery, a thrill over the anterior wall of the thorax, and as a result of the right ventricular hypertrophy, prominence of the upper part of the sternum. The symptoms of impaired pulmonic circulation are prominent, including cyanosis, dyspnœa, œdema of the lungs, and general anasarca. The majority of the patients die in childhood, but cases have been known to live until fairly advanced life.

Tricuspid Systolic Murmur.—A systolic murmur audible in the tricuspid area may be produced by

a. Regurgitation at the tricuspid orifice.
b. Anæmia.
c. Neuro-cardiac disease.
d. The puerperal state.

(*a*) **Tricuspid Regurgitation.**—The murmur of tricuspid regurgitation is systolic in time, with its point of maximum intensity at the lower end of the sternum, and extending outwards to the left for about an inch from the sternal border. This may be stated as invariably the location of the murmur when dependent upon pure tricuspid regurgitation; at most, it will not rise higher than the third left cartilage, nor extend outward beyond the position of the right ventricle. If it does do so, there must be considerable inflammatory thickening or alteration in the position of the heart. Associated conditions, which are very liable to exist, lead to departures from the above-mentioned conventional tricuspid area. Sometimes the murmur is most intense below the sternum; sometimes it is audible only at the apex. Under either of these circumstances the diagnosis is based upon the absence of signs which one expects in case of the presence of the other lesions thus suggested; *e.g.*, in case of systolic apex murmur, the abnormal sound will not be transmitted to the axilla or angle of the left scapula.

Under any circumstances the diagnosis of tricuspid insufficiency is not an easy matter. Of all cardiac murmurs it is the one most frequently overlooked. The associated signs, when present, are, however, unmistakable in their significance. Especially does this remark apply to venous and hepatic pulsations. The venous pulsation is best observed in the jugular veins, which may be so marked as to resemble carotid pulsation. It is much more readily extinguished by finger-pressure than is the latter; the veins of the neck are distended, ofttimes to double their normal size; when emptied by pressure, they will fill from below. The mistake must not be made of assuming that jugular pulsation is necessarily dependent upon tricuspid regurgitation. For this, such venous pulsation must be systolic in time. When presystolic, it is dependent upon contractions of the right auricle, and amounts only to a mere rhythmical undulation or distention.

Enlargement of the liver, with pulsations, is frequently present. This condition may be detected by placing one hand anteriorly and the other posteriorly over the liver, and practising firm palpation.

The consequences of tricuspid regurgitation are best studied by observing the condition of the right heart and the venous system. The right ventricle undergoes marked dilatation, as shown by heaving pulsation of the right ventricle felt at the epigastrium, increased cardiac dullness to the left, and undulating pulsation to the right of the sternum, general cyanosis, and ofttimes dropsy.

Tricuspid insufficiency is a rare lesion as the result of rheumatic endocarditis in adult life. It is apt to occur in the endocarditis of fœtal life and infancy. Malignant or septic endocarditis is also far more likely than the simple rheumatic variety to attack the tricuspid valves. Indeed, the involvement of the tricuspid orifice is strong presumptive evidence of the septic origin of an attack of acute endocardial inflammation.

Tricuspid regurgitation not infrequently occurs in old valvular affections with pulmonary engorgements. The first result of this complication is to relieve the distended pulmonary system. Hence tricuspid regurgitation occurring thus has been referred to as a "safety-valve action." While it thus relieves the passively-congested lungs, and with it many of the symptoms, it finally produces most disastrous consequences on the general circulation; hence it cannot be regarded as a conservative lesion.

(*b*) **Anæmic Murmurs** and (*c*) **Neuro-Cardiac Disease.**—Parrot, who first directed attention to the occurrence of tricuspid systolic murmur in anæmia, considered that actual regurgitation at that orifice existed, being dependent upon relative incompetence such as may occur at the mitral orifice under similar conditions. Balfour contends that this murmur is really of mitral origin, but exhibits an anomalous direction of transmission. Any danger of diagnostic error from this source is removed by the absence of venous pulsation, dilatation of the right heart, and cyanosis, in anæmic cases.

(*d*) **Tricuspid Murmurs During the Puerperal Period.**—Money has described a soft, blowing murmur, fairly long in duration, occurring in women during the lying-in period. In the majority of cases it was tricuspid in location, and systolic in time. Its precise nature has not been explained.

Tricuspid regurgitation undoubtedly occurs in some cases of acute dilatation of the heart from over-exertion, but under such circumstances is now usually attended by a murmur.

Tricuspid Presystolic Murmur.—A presystolic murmur with maximum intensity in the tricuspid area indicates the presence of tricuspid stenosis. Such a lesion is rarely diagnosed, and for several reasons. In the first place, it is of rare occurrence, and even then it exists in association with mitral stenosis. Hence its murmur is lost or escapes recognition in the presence of the mitral presystolic murmur. To differentiate these two murmurs when simultaneously present, the observer hears each in its conventional area, presenting fairly well-defined differences in pitch, intensity, and quality.

Pulmonary Systolic Murmurs.—The murmurs audible over the pulmonary area offer an interesting subject for study, owing to their variety and the uncertain value of the explanations offered for their existence. Guiteras has not inaptly referred to the pulmonary area as "the region of romance." Pulmonary systolic murmurs may be present in

a. Pulmonary stenosis.
b. Anæmia and chlorosis.
c. Uncovering of the conus arteriosis.
d. Mitral regurgitation.
e. Aneurysm.
f. Roughening of the aortic arch.
g. Obstruction of the pulmonary artery by embolism or thrombosis.
h. Neuro-cardiac disorders, of which exophthalmic goitre may be accepted as the type.
i. Displacements of the heart.
j. Structural disease in the pulmonary artery itself.

(*a*) **Pulmonary Stenosis.**—This is probably the rarest of all the causes of pulmonary systolic murmur, because pulmonary systolic murmur is a lesion of uncommon occurrence. The vast majority of cases are observed in infants, as the result either of fœtal endocarditis or of maldevelopment. Pulmonary stenosis acquired during adult life is apt to be the result of septic endocarditis, in which case the other valves of the heart also suffer; of atheroma; or of a direct blow over the heart, injuring the right ventricle and the pulmonary artery by reason of their superficial position.

When dependent upon stenosis at the pulmonary orifice, the murmur is situated in the conventional pulmonic area, *i.e.*, over the second left interspace near the sternum. It is often associated with a thrill which is most marked over the area of greatest audibility. The murmur is commonly rude and harsh ; exceptionally it may be soft ; it gives the impression of being generated close to the ear, and is increased when the patient assumes the recumbent position. It undergoes certain changes during respiration, which changes are not, however, diagnostic. Thus, it is increased on holding the breath, though it is afterwards diminished ; it diminishes almost entirely on forced expiration, and increases in intensity with energetic breathing.

Pulmonary stenosis occurring at any time of life is associated with hypertrophy of the right ventricle. Cyanosis is also a prominent feature, especially in infants and children.

(*b*) **Anæmia and Chlorosis.**—The systolic pulmonic murmur of anæmia and chlorosis is not associated with the usually observed concomitants of pulmonic stenosis, namely, the thrill, the hypertrophy of the right side of the heart, cyanosis, and dyspnœa. On the other hand, there are the characteristic blood changes of these conditions to point to the origin of the murmur. It is the most commonly observed of all pulmonic murmurs. It is usually soft and blowing in character ; occasionally it may simulate the organic type of murmur in this locality, and be rough and harsh. Like the organic murmur, it is influenced by variations in respiration. It may be associated with the well-known venous hum in the neck, or with murmurs in both carotid arteries. Their presence adds greatly to the certainty of the diagnosis.

(*c*) **Uncovering of the Conus Arteriosus Through Lack of Expansion of the Lung.**—The pulmonic systolic murmur from this cause is not associated with cyanosis, thrill, dyspnœa, or hypertrophy of the right ventricle. The murmur usually disappears when the patient holds his breath in deep inspiration. It is observed especially in the young of both sexes, and commonly disappears in adult life.

(*d*) **Mitral Regurgitation.**—This valvular defect is sometimes attended by a systolic murmur in the pulmonic region, in which case the true nature is determined by the concurrent signs of mitral insufficiency, namely, transmission of the murmur to the axilla or angle of the left scapula, and accentuation of the pulmonic second sound.

(*e*) **Aneurysm.**—This may present the murmur and thrill in the conventional locality ; but the presence of pulsation, the physical signs of a tumor and the pressure-symptoms will establish the diagnosis.

(*f*) **Roughening of the Aortic Arch.**—A murmur originating in this cause will be audible in the carotids, and there is no change in the size of the right ventricle. It occurs in persons who have reached the degenerative period of life.

(*g*) **Obstruction of the Pulmonary Artery by Embolism or Thrombosis.**—The incidence of a pulmonic systolic murmur from this cause is stated on the authority of Walshe, although others have failed to confirm him. The diagnosis of this condition will depend upon the associated symptoms. There will be a history showing the presence of an illness capable of producing embolism or thrombosis. The obstruction of the pulmonary artery will be characterized by suddenly-appearing dyspnœa, intense anguish, shallow breathing, cyanosis or pallor, coldness of the surface of the body generally, and, if death does not promptly follow, cough with hæmoptysis.

(*h*) **Neuro-Cardiac Disorders.**—The pulmonary artery is a favorable site for the murmurs in exophthalmic goitre and kindred disorders. The diagnosis must be based upon the associated symptoms, *e.g.*, goitre, exophthalmos, tachycardia, and the neurotic disturbances. It is fair to presume that in many of these cases there is some degree of dilatation, which, however, is incapable of recognition by the physical signs.

(*i*) **Displacements of the Heart.**—The pulmonic systolic murmur has been observed in cases of great displacement of the heart by pleuritic effusion, but which disappears after paracentesis. A similar murmur has been observed in the course of pulmonary tuberculosis, due probably to cardiac displacement by the contraction of fibrous bands. It should be remembered that marked displacement of the heart is not necessarily productive of this murmur.

(*j*) **During the Puerperium.**—The pulmonic systolic murmurs not infrequently observed during the lying-in period are of comparatively short duration, disappearing within a few days.

Pulmonary Diastolic Murmur.—A diastolic murmur in the pulmonic area occurs as a result of regurgitation at the pulmonary orifice.

Pulmonary Regurgitation.—While the signs and symptoms of pulmonary regurgitation are clearly defined, and the diagnosis of the affection should be readily made, it is a fact, nevertheless, that it commonly fails of recognition during life. The majority of cases are observed during early adult life. It is often of congenital origin, but in that case it is usually associated with pulmonary stenosis. As an acquired lesion, it is due to rheumatic endocarditis, the eruptive disorders, and less rarely to degenerative processes. The murmur is diastolic in time, and has its maximum audibility in the second left interspace. It is transmitted *towards* the apex of the heart, and along the left of the sternum. It is sometimes associated with a diastolic thrill felt over the pulmonic area. The presence of hypertrophy and dilatation of the right ventricle is shown by increased cardiac dullness to both the right and left of the sternum. Venous stasis is a necessary concomitant, and results in enlargement of the abdominal viscera, catarrhs of the mucous membranes, and serous effusions. Dyspnœa and cyanosis, though commonly present, are not, excepting in the terminal stages, apt to be prominent features when the patient is at rest.

Pulmonary stenosis may readily be confounded with aortic regurgitation, but a correct diagnosis should be made, excepting in those instances in which the two lesions co-exist. As already stated, the murmur of aortic regurgitation is heard with maximum intensity to the left of the sternum, herein constituting an important reason for diagnostic errors. The pulmonic regurgitant murmur is not transmitted directly to the apex, while the aortic may be heard distinctly at that point. The concurrent symptoms of aortic regurgitation, namely, the Corrigan pulse, the remarkable degree of cardiac hypertrophy, and the capillary pulse, make the diagnosis of that lesion clear.

When aortic and pulmonary incompetence co-exist, a correct diagnosis is frequently impossible. There is no difficulty in recognizing the aortic incompetence in such cases; but the pulmonary-valve condition can only be inferred by the associated symptoms and the history of the case.

Thus far, my review of the investigation of cardiac diseases has been limited to a consideration of the physical signs. It is now in order to treat of the

Subjective Symptoms.

The first of these is

Pain.—It is well to bear in mind that præcordial pain may be associated with any variety of cardiac disease, some, however, with much greater frequency than others. It is equally true that pain is not a necessary concomitant of any disease of the heart. The majority of cases of pain in and about the præcordia have other than a cardiac origin, and include such affections as intercostal neuralgia, myalgia, chlorosis, gastro-intestinal disturbance, pleurisy, and actual disease of the thoracic walls, *e.g.*, periostitis of the ribs. Each of these will be found thoroughly elaborated elsewhere; so, for the present, præcordial pain originating in cardiac disease will alone be considered.

When investigating a case of alleged cardiac pain, it is important that the physician obtain an accurate idea as to its location, both as to the point of maximum severity and the area over which it radiates; its relation to rest and exertion; to the taking of food in general and of particular kinds of food, and to the time of day. If this is not done, the correct origin of the pain cannot be ascertained.

Cardiac pain may present all degrees of severity and constancy. In many instances it consists of but a dull aching; in others it is of the most agonizing severity. In any event it is liable to be attended by a peculiar anxiety or sense of impending dissolution, which usually depicts itself on the face of the patient by an anxious or terror-stricken expression.

The affections of the heart and blood-vessels which may be attended by pain include the following:

a. Pericarditis.
b. Pneumo-pericardium.

c. Acute aortitis.
d. Subacute and chronic aortitis.
e. Aneurysm of the aorta.
f. Endocarditis.
g. Valvular diseases of the heart.
h. Angina pectoris.
i. Myocarditis.
j. Rupture of the heart.

(*a*) **Pain in Pericarditis.**—Pain is a very common phenomenon of acute pericarditis,—much more so, indeed, than in myo- or endocardial inflammation. Its severity varies in individual cases; thus it may be dull or so slight as to escape more than casual notice on the part of the patient; in young children it is often entirely absent. Or it may be of agonizing severity, and radiate over the entire chest and extend down the arms, especially the left. In the majority of cases it is scarcely more than annoying. It is aggravated by pressure and movements. There may be only a mere tenderness without spontaneous pain; indeed, the superficial hyperæsthesia may be remarkable. Still another location for pericarditic pain is deep in the chest, between the shoulder-blades. Sometimes pain from this cause is referred to the epigastrium, and appears later in the course of the disease than pain over the heart. The diagnosis is based upon the history of rheumatism, the constitutional symptoms of inflammation, and the discovery of the pericardial friction-sound.

(*b*) **Pneumo-Pericardium.**—This lesion implies an opening into the pericardium so arranged as to connect with an air-containing organ. The onset of symptoms must be sudden, among the first being severe pain, which is associated with intense anxiety, dyspnœa, cyanosis, and collapse. The concurrent signs which establish the diagnosis include the history of the case as having a bearing upon its etiology, protrusion of the chest-wall when the quantity of pericardial air is extreme, and tympanitic resonance replacing in part the normal præcordial percussion-dullness. If death does not result promptly, pericarditis will develop, and then we have the signs and symptoms of that disorder.

(*c*) **Acute Aortitis.**—The pain in this disease is of the most agonizing character. Originating in the aorta, it is referred to beneath the entire extent of the sternum and along the vertebral column posteriorly. It is associated with great anxiety and distress. Sometimes it radiates over the chest and down one or both arms; sometimes there is a sensation of constriction about the mid-sternal region, or a feeling as of a lump in the throat simulating the globus hystericus. Associated symptoms include a pale or yellowish-clayey hue of the face, dyspepsia with nausea and eructations, anxious expression of countenance, and evidences of dilatation of the aorta. Under the symptom last mentioned may be included an upward displacement of the subclavian arteries, especially the right, so that their pulsations may be visible above the sternum, and increased percussion-dullness, showing enlarged outlines of the aorta. There may also be disturbance of the normal aortic sounds. The history usually shows the pre-existence of a cause in some infection, notably typhoid fever, influenza, malaria, syphilis or septic infection.

(*d*) **Chronic Aortitis (Atheroma).**—The pain in these cases is of the character usually dependent upon cardiac disorders. It occurs in paroxysms, and is often associated with disease of the aortic valves. It is especially liable to occur in gouty, syphilitic and alcoholic subjects.

(*e*) **Aneurysm of the Aorta.**—Pain in aneurysm of the aorta may be dependent upon the aneurysm, *per se*, or upon pressure by the same upon the sternum or vertebræ or upon adjacent nerves. Pain due strictly to the aneurysm is of a dull, aching character, and is aggravated by any movement.

Pressure of the tumor upon bony structures, *i.e.*, upon the sternum and bodies of the vertebræ, leads to their erosion, and is accompanied by a constant boring or gnawing pain, more or less sharply localized.

The most severe pains of aneurysm, however, are those originating in pressure of the tumor upon sensory nerves. Such pains are remarkable for their acuteness, their paroxysmal exacerbations and remissions, and for the wide extent over which they may radiate. Thus the pain, while centreing in the præcordia, may also be manifest in the neck, shoulders, both arms, and even in the face. It has well been stated that these pains of aneurysm closely simulate those of angina pectoris.

So far as the situation and the character of the pains are concerned, there is nothing in either that may be regarded as diagnostic. Nevertheless, pain, although often absent in thoracic aneurysm, should lead the physician to examine the patient carefully for the physical signs of that lesion.

Pain is also of importance from a prognostic standpoint, and, as was wisely observed by Walshe, it is an important element in determining early emaciation.

(*f*) **Endocarditis.**—While pain is not considered an important diagnostic symptom of endocarditis, nevertheless it is a not uncommon signal-symptom of the disease, in that it is often the first phenomenon directing attention to endocarditis as a complication, *e.g.*, of inflammatory rheumatism. It is associated with dyspnœa, fresh accession of fever, excited action of the heart, etc. Auscultation will discover the presence of murmurs, or murmurs additional to those already existing. In simple endocarditis the fever is not of high grade. It is otherwise with the malignant variety, in which the temperature assumes the characteristic remittent type of septic infection.

(*g*) **Valvular Affections of the Heart.**—Pain has no diagnostic value in valvular diseases, as it may be present in any variety. It is, however, by far more commonly observed in affections of the aortic orifice than in all the others collectively.

(*h*) **Angina Pectoris.**—It is with angina pectoris that the characteristic præcordial paroxysmal pain is most intimately associated in the professional mind; so much so, in fact, that there is altogether too strong a tendency to diagnose this disease, or, more properly speaking, "syndrome," when another condition is present, basing the conclusion solely upon the presence of pain. In angina pectoris the paroxysm is of sudden onset; the pains are of the most atrocious character, centreing about the heart, and extending thence into the neck and down the arms. They are associated also with a feeling as if the heart was held in a vise or grasped by a hand, and with the most intense anguish. The face becomes pale or of an ashy hue, the extremities become cold, and respiration is embarrassed, but there is no dyspnœa. In a short time, although it may seem hours, the paroxysm ceases, the patient breaks out into a cold sweat, and is left in a condition of profound prostration. The pulse may exhibit no abnormality, while in other cases it may be one of inordinately high tension. Exceptional cases are observed in which the pain radiates into very unusual localities, *e.g.*, to the scapulæ, back of the neck and occiput; or still more exceptionally into the pelvis, and even into the lower extremities.

In some instances the onset is so sudden and the attack so severe that a fatal termination takes place before there is an opportunity for the pain to appear (*angina sine dolore*).

The main difficulty in the diagnosis of angina pectoris is that of separating it from the hysterical disorder,—*pseudo-angina*. The latter occurs in females at the age favorable to the incidence of hysterical seizures generally. The paroxysm is determined by the ordinary causes of hysterical paroxysms, *e.g.*, emotional. The constitutional predisposing causes include those of debilitating character, as anæmia, lactation, overwork, long-continued worry, etc. Throughout the attack the patient may perform movements of a most evident hysterical character; thus with the severity of the pain she wrings her hands, clutches at her hair, or grasps the hands of a bystander. There may even be hysterical mental manifestations, such as special sense-delusions, fears of this or that danger, but never the peculiar anguish or terror which one observes in true angina pectoris, and which, once seen, is never forgotten.

Nor should angina pectoris be confounded with cardiac neuroses dependent upon the excessive use of tobacco or alcohol. The age of the patient and the presence of other symptoms traceable to this addiction should guard against error.

Some cases of angina pectoris in which the vaso-motor symptoms predominate, indeed are far more obtrusive than the pain, may escape recognition. Nevertheless, even in these some of the characteristic anxiety and præcordial anguish will be present. The vaso-motor symptoms present usually include pallor of the arms almost to the extent of a local syncope, sometimes associated with coldness, stiffness, formication, and anæsthesia; or the reverse condition, local asphyxia, may obtain, and the arm exhibit swelling, with purplish or bluish discoloration. Attacks of this character are readily produced by slight exposure to cold, even the washing of the hands in cold water being capable, in the predisposed, of provoking a paroxysm.

(*i*) **Myocarditis.**—The ordinary pain incident to chronic myocarditis is rarely more than a sense of uneasiness, distress or anxiety. Such a symptom is common very early in the course of the affection, before the cardiac weakness has become pronounced. There may be an associated irritability of the heart, as evidenced by palpitation and tachycardia, under slight physical and emotional provocations. It may be that the distress arising from these latter symptoms occasions as much suffering as actual pain. It is not uncommon for the early stages of myocarditis to exhibit attacks of intercostal neuralgia. The diagnosis is based, of course, upon the physical signs of weakened myocardium.

(*j*) **Rupture of the Heart; Hæmopericardium.**—This accident is manifested clinically by the sudden incidence of pain in association with pallor, prostration, and collapse. The patient dies shortly from shock. In some few cases life is prolonged for a few hours, in which case dyspnœa and cyanosis are prominent clinical features.

Palpitation.—By this term is meant a rapid and forcible action of the heart, which makes itself perceptible to the patient and occasions considerable distress and suffering thereby. This symptom may be regarded as physiological when it results from prolonged or excessive muscular exertion, in which case, notwithstanding the subjective sensations making the cardiac activity evident to the patient, there is no distress.

The attendant phenomena of palpitation will vary greatly according to the severity of the case. Thus, in mild ones, there may be but a slight sensation of fluttering experienced about the præcordia, associated possibly with a slight sense of sinking at the epigastrium. In the more severe examples, the throbbing sensations will be experienced in the blood-vessels throughout the body, especially, however, those of the chest and head. In such cases, moreover, respiration is apt to be embarrassed, being sighing or irregular, but usually without dyspnœa. Sometimes there are anxiety and sense of constriction, tinnitus aurium, headache, vertigo, general nervous agitation, and tremor. Vaso-motor symptoms may attend, as exhibited by flushing and sweats.

Palpitation is a paroxysmal symptom, the attacks of which usually follow some cause, generally reflex disturbance by reason of disease in some other organ, or psychical impressions, especially those of a depressing character. Those cases which arise from slight physical exertion, *i.e.*, such as would not be operative in a healthy individual, are ordinarily dependent upon organic disease of the heart. Each individual case seems to have its special etiological factors, which vary but little from time to time.

As already stated, palpitation is but a symptom, and in no sense a disease. It may result from disease of the heart, or from affections of other portions of the body,—in the great majority of cases from the latter. There is no organic cardiac affection which may not be attended by it, but in such cases the other evidences of cardiac failure are so prominent that the palpitation scarcely engages the attention of the patient. The physical signs of cardiac disease, as murmurs, hypertrophy and dilatation, establish the diagnosis.

It is the cases originating in the so-called functional diseases of the nervous system, however, that give the physician the most trouble. Such cases are often spoken of as cardiac neurasthenia. The patient permits his mind to dwell on the cardiac distress, conjuring up all possible horrible contingencies as the result of his illness. Back of this condition of the nervous system will usually be found the ordinary causes of neurasthenia, *e.g.*, worry, grief, misfortune, hardships, overwork, or other depressing agencies, sexual excesses, anæmia, indigestion (gastric neuroses), sexual neuroses, utero-ovarian disease, etc. Among these neurasthenic cases should be included that troublesome class of palpitations occurring at puberty and during the climacteric. The neurotic foundation is necessary to the incidence of the palpitation, however apparent may be the disease in adjacent viscera.

Other cases seem to have a more truly reflex origin, the paroxysms evidently resulting from disease of the stomach or pulmonary affections. It may be that the palpitation may ensue upon distention of the stomach by a heavy meal, by the partaking of a particular article of diet which habitually disagrees, or by distention of the stomach by flatulence.

Again, we may observe cases originating in toxic causes,—poisons produced within the system, as lithæmia, gout, and the toxins originating by gastro-intestinal indigestion. Very prolific causes of palpitation are found in excessive use of alcohol, tobacco and coffee.

Other symptoms of heart disease than the two above described in detail include difficulty of breathing (dyspnœa), dropsy, venous congestion of the liver leading to enlargement of that organ, congestion of the lungs, cough, diminished secretion of urine, albuminuria, indigestion, and mental sluggishness. All of these symptoms may be studied more systematically in connection with a consideration of the other phenomena of disease of the various viscera to which they are more directly referred.

Sudden Death in Heart Diseases.—It is important that the victims of heart diseases should have the commonly-held apprehensions concerning the liability to sudden death corrected. While any case of disease of the heart may have this unfortunate termination, it is only in a comparatively small proportion, and these distributed among few pathological lesions, that a sudden termination is to be apprehended. Among the valvular diseases, those of the aortic orifice are the only ones deserving serious consideration in this respect. Lesions at other orifices may terminate with unexpected suddenness; but in each case of this character it will be found that there will appear numerous symptoms, giving due warning of an approaching fatal issue,—*i.e.*, the sudden death will be preceded by symptoms of ruptured compensation.

Among other than valvular lesions, sudden death is to be feared in angina pectoris, chronic myocardial degenerations, and the acute nervous and myocardial lesions complicating the infectious diseases, *e.g.*, diphtheria and typhoid fever, and obstructions of the coronary arteries. In angina pectoris, death is usually preceded by the præcordial pain and other symptoms diagnostic of this disorder, and, too, only after the patient has suffered from other attacks. Exceptionally, however, death succeeds a first seizure; and in still other instances the fatal issue succeeds the advent of the symptoms so quickly that there is no time for the appearance of pain (*angina sine dolore*).

In cases of suddenly fatal myocardial degeneration, it will usually be observed that the unfortunate result is often brought about by the incidence of causes throwing increased work upon a greatly-weakened heart-muscle. Thus, the running for a train, sudden exposure to an intensely cold atmosphere, especially after having been in a warm room, overloading the stomach, etc., may constitute important exciting causes. In many of these cases there will be found, if carefully sought, evidence of a laboring right ventricle to explain the serious condition of the patient.

Aortic aneurysm may have a suddenly fatal termination owing to rupture of the sac. This may be preceded by profuse hæmoptysis, or the hæmorrhage may be concealed, and the symptoms of collapse only present themselves.

If all cases of heart disease are carefully studied, the clinician can readily determine, with but few exceptions, those which are liable to terminate in a sudden fatality. In other words, careful study will demonstrate that sudden death from heart disease is seldom unexpected.

Resume of the Symptomatology of Such Diseases of the Heart and Aorta as Have Not Been Sufficiently Explained in the Foregoing Pages.

1. Aneurysm of the Aorta.

Aneurysm of the aorta may be either fusiform or sacculated. It may involve either the ascending, transverse or descending portion of the vessel, or it may be limited to the intra- or extra-pericardial portion of the ascending aorta. In each locality special symptoms or clinical signs may be produced, ofttimes leading to an exact topographical and pathological diagnosis, and yet, in too many instances, so obscure as to enshroud the recognition of aortic aneurysm in the greatest obscurity.

The diagnosis of aneurysm of the aorta depends upon the recognition of a tumor exhibiting pulsatile expansion in the area of the chest occupied by the aorta. We expect dullness on percussion, with pulsation within that area of dullness. Palpation, moreover, detects a systolic thrill in probably less than half the cases. Auscultation sometimes discovers a systolic murmur over the area of dullness. This murmur is so frequently absent in the course of aortic aneurysm, and is so frequently present in other conditions, that it is not to be regarded as a valuable clinical sign. In addition to these symptoms, there may appear any of the following: Inequality of the pupils, inequality in the radial pulses, severe pain in the præcordium, radiating into the left arm or neck, and œdema of one arm.

The Tumor.—The presence of the tumor is largely dependent upon the relation of the aneurysm to the walls of the chest; thus, in cases of aneurysm of the ascending aorta, it exerts pressure early in its course upon the right margin of the sternum, eroding that bone, and leading to its appearance externally at the level of the second or third costal cartilages. This tumor is hard; indeed, it may be as hard as any solid growth. From the latter, however, it is readily differentiated by its expansile pulsation. Solid tumors lying over the aorta may present an apparent pulsation, *i.e.*, the rise and fall with the dilatation and contraction of the underlying vessel, but there is never the expansile pulsation observed in aneurysm.

The Pulsation.—The pulsation, as already stated, is distinctly expansile. It may be detected by both inspection and palpation. In the early stages of the disease, when it is illy-defined, it may be detected by placing the patient so that the light falls obliquely upon his chest, while the observer has his eyes on a level with the suspicious locality.

The Thrill.—The thrill of aortic aneurysm is systolic in time and vibratile in character. It is often absent, especially in cases in which the tumor is filled with a large lamellated clot. It is especially apt to be present in fusiform aneurysms.

Tracheal Tugging.—The method of eliciting this symptom has been described elsewhere. Tracheal tugging is almost certainly diagnostic of aneurysm of the aorta. Its absence, however, proves nothing, as it is not invariably present in the disease under consideration. Care should be observed not to confuse it with a pulsation transmitted from the vessels of the neck, tracheal tugging being, as it name signifies, "a tugging by the trachea" in response to the pulsatile retractions of the aneurysmal walls. A true tracheal "tug" may occasionally be observed during inspiration in healthy persons, and must not be accepted as of pathological import.

Percussion-Dullness.—The presence of dullness on percussion will depend upon the location of the aneurysm. When the latter is deeply situated, this valuable sign will be absent. When the tumor lies directly beneath the thoracic wall, the dullness will usually manifest itself early, and be situated over the upper end of the sternum and the second and third right intercostal spaces. Necessarily, the location of the dullness will vary with the position and size of the growth.

The Murmur.—This symptom is so frequently absent, and of so uncertain a character, that it can hardly be regarded as of much importance in the diagnosis of aortic aneurysm. I take the trouble to refer to it here, in order to correct prevalent ideas as to its value.

So much for the clinical signs of aneurysm. The pressure-symptoms include pain, laryngeal paralyses, inequality of the pupils, pulmonary symptoms, and inequality of the radial pulses. Each of these symptoms has been or will be considered elsewhere, and the reader is referred to such paragraphs for their study.

According to the situation of the aneurysm, symptoms of this or that character predominate. Thus, in aneurysm of the ascending aorta and first portion of the arch, the physical signs are pronounced. Hence we speak of the disease in this situation as the aneurysm of physical signs, in contradistinction to the lesion involving the last portion of the arch, and the descending aorta, which may be described as the aneurysm of pressure-symptoms.

Aneurysm of the Ascending Aorta.—The special symptoms of aneurysm of the ascending aorta include the following: (a) Pulsating tumor situated beneath the second and third right costal cartilages and the upper end of the sternum, which will, according to the direction in which it exerts pressure, compress the superior vena cava, the upper lobe of the right lung or the right bronchus, the right recurrent laryngeal nerve, the pulmonary artery, the heart itself, or the inferior vena cava. In a small percentage of cases the aneurysmal tumor is found to the left of the sternum.

Aneurysm of the Transverse Portion of the Arch of the Aorta.—Pressure-symptoms are prominent in aneurysm of the transverse portion of the arch. Owing to the relatively small diameter of the chest in this region, pain is an early feature. Pressure upon the trachea leads to interference with respiration as exhibited by stridulous breathing. Sometimes, however, the pressure is upon the left bronchus, which condition is made evident by deficient respiratory murmur on that side and lack of expansion of the left lung. Dysphagia is sometimes present, and is due to pressure of the tumor upon the œsophagus. Tracheal tugging is an especially important sign of aneurysm in this locality. Laryngeal symptoms are also prone to be present, asserting themselves as a loud, clanging, brassy cough, or a disorder of phonation from paralysis of the left recurrent laryngeal nerve. The latter may be present without any disturbance of function discoverable apart from the laryngeal examination. Cases have been reported in which this recurrent laryngeal paralysis constituted for a long time the only symptom suggestive of the presence of aneurysm.

Aneurysm of the Descending Portion of the Arch.—In aneurysm of the descending portion of the arch the pain arises from pressure upon the intercostal nerves and from erosion of the bodies of the vertebræ. Hence it assumes the type of intercostal neuralgia in the one case, and is of a dull, boring character near the spine in one or both interscapular regions. Physical examination may show a pulsatile tumor posteriorly in the left interscapular region, or, in its absence, a dullness on percussion in the same locality.

Differential Diagnosis of Aortic Aneurysm.—Aortic aneurysm is sometimes supposed to exist when the condition present is simply a misplacement of the aorta resulting from a deformity of the thorax, usually of rachitic origin. Scoliosis is always present in such cases. The diagnosis in such cases is based upon the absence of any abnormal percussion-dullness other than that arising from visceral displacement, the absence of pain and other pressure-symptoms of aneurysm, and the normal character of the aortic sound.

Retraction of the lung may give rise to visible or palpable pulsation to the right of the sternum; but such palpation is not expansile, and there is no increase in percussion-dullness to the right of the sternum. There are present, moreover, the physical signs indicative of the pulmonary disorder producing said retraction.

Visible and palpable pulsation may be observed to the left of the sternum, owing to fibroid disease of the left lung uncovering the pulmonary artery. The absence of an accentuated aortic second sound and pressure-symptoms, and the physical signs of pulmonary fibrosis, establish the diagnosis.

2. Angina Pectoris.

The clinical study of angina pectoris has been very much hampered by the numerous attempts of authorities to differentiate a true and a false variety of this so-called disease. Inasmuch as the complexus of symptoms included under the name of angina pectoris may, as is universally acknowledged, be brought about by a variety of pathological causes, of which calcification of the coronary arteries is the chief, it is best to study the condition as a symptom complex, and forget that there ever has been an attempt made to differentiate the alleged true and false varieties. The importance of these preliminary remarks is manifest to any one who has studied the many authorities on cardiac diseases and has, in the confusion of ideas therein presented, endeavored to reconcile the diversity of doctrines taught. To me, the writings of Gibson approach the truth more closely than do those of any other author.

The essential feature of angina pectoris is its paroxysmal nature, and its principal symptom is the præcordial pain radiating over the chest and even down the arms, associated with a sense of impending death. It is a mistake, however, to regard the pain as always of severe character. Such it is in many, perhaps the majority, of instances. To say that it is always severe is placing too great limitations upon our study of the subject from a practical standpoint. It has been my misfortune to have seen a number of cases, all ending in sudden death, in which the paroxysms were associated with a sense of constriction about the heart and more or less anguish, brought on by exertion ofttimes but slight, the patient enjoying comparative comfort during the interparoxysmal period. The intensity of suffering in individual instances presented all grades of severity, so that it is fair to assume that symptomatic differences originated not in variety of pathological conditions so much as in differences of temperament and the progress of the disease.

The sense of impending dissolution seems to be as important a symptom as the præcordial pain. Indeed, from a diagnostic standpoint it may be more so. Certain it is that no condition manifests it more strikingly than does angina pectoris.

While the respirations are shallow during the attack, there is no difficulty of breathing, for the patient can, on request, take a full inspiration. The respiratory disturbance seems to be produced by the fear that free efforts at breathing will aggravate the pain.

The countenance of the patient is consistent with the mental anxiety and sense of impending death. To use the words of Gibson, "it usually betokens unutterable anguish and fearful anticipation."

There is no characteristic state of the pulse in angina pectoris. Attempts to differentiate true from false angina pectoris have been based upon this symptom : When pulse-tension is found to be high, it is assumed that the attack has been brought about by this circulatory condition ; the increased vascular pressure is assumed to be the primary condition, the angina pectoris being a result. A careful study of cases of angina pectoris will show that most of the patients have arterio-sclerosis in some degre and of this, increased vascular pressure is a common attendant. And we find high tension the usual condition of the pulse during the paroxysm Nevertheless, there are many cases in which the pulse is weak, irregular, rapid.

The position assumed by the patient will vary according to the ci cumstances attending the seizure. If taken while standing or walking, speedily seeks a place for support; if in bed, he sits up, with body be forward.

In describing the so-called pseudo angina pectoris, it seems to me th the acceptance of the four varieties of this condition as tabulated by G son will greatly simplify the clinical study of the subject.

(*a*) **Reflex angina pectoris,** in which the attacks are precipitated some irritation, generally in the gastro-intestinal tract, though they m also arise from pulmonary and nervous conditions. In nearly all such cas careful examination will discover pronounced evidences of myocardial a arterial degeneration.

(*b*) **The vaso-motor angina pectoris of Nothnagel,** characteri by " sudden onset of coldness, or lividity of the extremities, often associa with profound perspiration and attended by severe cardiac pain, along w palpitation and feelings of faintness." Those cases, likewise, are prone present clinical evidence of cardio-vascular degeneration.

(*c*) **Neurasthenic angina pectoris,** which is really not angina p toris in any sense of the term, but may well be mentioned here from standpoint of practical diagnosis. The pain is often severe, and there be a sensation of fluttering and faintness. But the temperamental peculi ties of the patient and the stigmata of neurasthenia render the diagn easy. It must not be forgotten, however, that neurasthenics, especially th of gouty or lithæmic type, may acquire angina pectoris as may the ordin individual.

(*d*) **Hysterical angina pectoris** is likewise not angina pect Cases receiving this designation are better termed mastodynia. The tient is usually a young woman. The paroxysm is brought about by s emotional cause. She clutches at the heart and cries energetically w out respiratory embarrassment; indeed, the very activity of her movem are inconsistent with the dread disease from which she is alleged to suffering.

Attacks of angina pectoris vary greatly in duration. In many stances the symptoms pass away within a few minutes, *i.e.*, as soon as exciting cause can be removed. The majority of cases do not conti more than five or ten minutes. Exceptionally, the symptoms last for eral hours. Death may take place almost immediately; usually, howe not until several attacks have appeared. The interparoxysmal stat health will vary. Authorities seem to teach that it is up to the standard observed in epilepsy, with which disease it has been compared. Careful clinical investigation, I am satisfied, will discover, in nearly all, the signs and symptoms of cardio-vascular degeneration.

Etiological factors have some slight bearing upon the diagnosis, although the clinical history of the disease is sufficiently distinctive. They include long-continued mental and physical strain, heredity, the degenerative period of life (after the age of fifty), sex (males being especially liable), gout, diabetes, alcohol, tobacco, tea, coffee (the latter two to but a slight extent), syphilis (by reason of its influence in producing arterial degeneration), and the infectious diseases, notably influenza and typhoid fever.

Some paroxysms appear without the intervention of an exciting cause. In the majority, some physical exertion or mental emotion, pleasant or otherwise, is the exciting factor. With the progress of the case it seems that slight causes become more readily operative.

3. Myocarditis.

In the following pages it is the intention of the author to include under the term of myocarditis all those numerous cases having cardiac weakness as a characteristic feature. From a pathological standpoint, such a course may be open to strong criticism; but inasmuch as the clinical features presented bear strong similarities,—so strong, indeed, as to make them indistinguishable at times,—it seems to have the merit of practicability. The various pathological conditions thus included are simple cardiac weakness, cardiac atrophy, fatty infiltration, fatty degeneration, and acute and chronic myocarditis.

Simple Cardiac Weakness.—This term is meant to convey the idea of a heart weak in function, yet possessing no discoverable alterations in structure. Many of these are examples of congenital inadequacy, the organ being abundantly able to perform its duties as long as no extra demand is made upon it. As soon, however, as such extra work is attempted, it yields, and, if relief is not soon obtained, undergoes dilatation. As an acquired condition, it may follow any of the causes which produce degenerative changes in the myocardium; and this fact suggests that the simple weak heart is, after all, but a mild example of one of the degenerative affections to be described hereafter. These causes include the specific infectious fevers, prolonged exertion, alcoholic excesses, the various anæmias, and inadequate nutrition.

The congenital cases are characterized clinically by their general systemic weakness. The acquired cases present, in addition to the debility produced by the primary disorder, well-marked breathlessness on slight exertion, and palpitation, sometimes associated with faintness, and even attacks of syncope. The state of the pulse well indicates that of the heart. There is evident tendency to venous stasis, especially in cold weather. Physic examination gives every evidence of impaired cardiac power.

Cardiac Atrophy.—By this term is meant a diminution in the weig of the heart by reason of atrophy of its muscular structure. There a congenital cases in which the muscle, as far as present, is normal in stru ture, but deficient in quantity. Most of the cases coming under this cat gory exhibit other evidences of impaired development, especially childis ness. "Infantilism" is a good general term to describe such cases. T acquired cases may result from syphilis, cancer, tuberculosis, diabetes, a other wasting diseases. Clinically, atrophy of the heart presents the sym toms of the primary affection with the physical signs attendant upon small heart with empty arteries and low blood-pressure. Care must observed lest the physical signs of pulmonary disease, as emphysema, ca a simulation of those produced by cardiac atrophy.

Fatty Infiltration of the Heart.—The etiological factors of fatty filtration of the heart include heredity, the degenerative period of life (a the age of forty-five years), lack of exercise and fresh air, excessive eati and the abuse of alcohol.

In patients suffering from this complaint the deposition of fat is limited to the region of the heart by any means, but is widespread. oppression of breathing from which they suffer is usually attributed by patient to his inordinate weight. There is frequently, as in other case cardiac insufficiency, faintness and vertigo. The heart is usually fo somewhat enlarged and feeble in its action. The liver is likewise enlar The digestive functions are usually good. The physician ordinarily fi considerable difficulty in determining the condition of the heart. The g deposit of fat in the thoracic walls makes the percussion-signs uncertain well as muffling the heart-sounds to auscultation. Notwithstanding the feeblement of heart-sounds thus occasioned, it will be noted that the sound especially is affected ; indeed, it may be replaced by systolic mur over both sides. Irregular action is sometimes observed, and there ma modifications of the Cheyne-Stokes type of breathing. With failing p of the heart may come œdema of the lungs, deficient urination, drowsi etc. The diagnosis depends upon a recognition of weak cardiac actio association with obesity.

Cases of fatty infiltration are progressive unless cured by suit treatment. It is possible for the condition to terminate suddenly in de

Fatty Degeneration of the Heart.—This is a far more serious dition than that above considered. Men are more likely than women t its victims, and hereditary predisposition is an important factor. It is frequently observed during the degenerative period of life, although have been reported as occurring in the very young. Sedentary habits alcoholic excesses are also frequent etiological factors. The determi causes of fatty degeneration are found in inadequate blood-supply and lessened work of the heart, the latter through sedentary habits. Under the former may be mentioned the many varieties of anæmia, the different cachectic disorders, including cancer, syphilis and tuberculosis, scurvy, gout, interstitial nephritis, etc., and many of the acute infections, as typhoid fever, diphtheria, variola, erysipelas, typhus, septicæmia, etc.

The symptoms of fatty degeneration of the heart are so poorly defined that it is impossible to diagnose the condition with certainty, although one may, as a rule, safely infer its presence under certain circumstances. The greatest difficulty is encountered in differentiating it from chronic myocarditis, the clinical phenomena of which are practically identical with those of fatty degeneration. The symptoms of cardiac insufficiency are usually pronounced. Unlike fatty infiltration, the digestive functions are impaired, the appetite being poor and dyspeptic symptoms present. The patient experiences uneasy sensations referred to the præcordia. It may be that these sensations amount to actual pain, and suggest the presence of a condition allied to angina pectoris. Sometimes morbid sensations are described as "flutters," "stammers," "stoppages," indicating the subjective equivalents of arrhythmia. There is no conventional pulse-characteristic in fatty degeneration, unless it be weakness. In the majority of cases it is rapid, and of low tension; in a minority, especially in cases running a very chronic course and in old subjects, it is abnormally slow. The apex-beat is barely, if at all, discernible by palpation. When present, it gives the impression of a short, feeble tap. Sometimes there is only a weak impulse felt at the lower end of the sternum at its left border. The outlines of the heart, as discovered by percussion, are not altered unless dilatation has set in. The first sound is greatly enfeebled; indeed, it may be barely discernible. The urine is generally greatly diminished in quantity, and may contain albumin and casts.

Fatty degeneration usually pursues a chronic course, progressing to complete cardiac failure. Sudden death is not uncommon under the stress of some slight exertion.

Acute Myocarditis.—This is always a secondary affection, resulting for the most part from constitutional or acute infectious diseases, as typhoid fever, diphtheria, septicæmia, or inflammatory rheumatism. The diagnosis of the affection is attended by the greatest uncertainty. One can only infer its presence when the symptoms of failing heart-action complicate one of the acute infections which are known to be capable of producing it. It usually runs a rapidly fatal course.

Chronic Myocarditis.—Under this term is now included a large class of affections resulting in increased connective-tissue formation in the myocardium, with degeneration of the muscular fibres. It is only within recent years that these cases have received careful clinical and pathological study, so that it cannot be said that we are in a position to make positiv diagnoses during the life of the patient. In a general way it may be state that the blood-supply of the myocardium is rendered deficient by reaso of endarteritis, thrombosis, embolism, etc., in the majority of cases. Mos of the examples of this disease are observed in men who have passe middle life, especially those whose hearts have been subjected to inordinat physical strain, as by excess in athletics and laborious occupations. Alcoh is an undoubted predisposing cause. Most cases are associated with generalized arterio-sclerosis; hence the disease is sometimes referred to a "gouty heart." Chronic toxæmiæ, notably lithæmia and syphilis, mu also be borne in mind as prolific causes.

The importance of syphilis in the production of chronic disease of th myocardium is not sufficiently appreciated. A careful study of the subje demonstrates that syphilis may cause myocarditis in three ways: (*a*) B the production of a chronic arteritis with secondary myocardial fibrosi (*b*) by the formation of gummatous tumors, which are, in turn, surround by connective-tissue formation; (*c*) and by a chronic diffuse myocarditi itself essentially of syphilitic nature.

The symptomatic features of the disease are not sufficiently chara teristic to make them a reliable guide in establishing a diagnosis. O must depend also upon the history of the case, the alleged etiologic factors, and the associated conditions. Among the earlier symptoms w be found, at times, a remarkable cardiac irritability, so that, under the infl ence of slight stimuli, pronounced degrees of tachycardia may superven The patient may complain of palpitations, flushes, breathlessness on slig exertion, and even anginoid seizures. There is usually abundant eviden of vascular changes. The temporal arteries are tortuous and have thic ened walls. The radial arteries are thickened, and there is increased vasc lar pressure. In the late stages of the disease the pulse-tension will found to be diminished. There is nearly always more or less cardiac dila tion. In the early stages of the disease the auscultatory signs are th usually attendant upon arterio-sclerosis; with diminution of tension and t advent of dilatation they become those of cardiac failure. The urine w nearly always show the evidences of chronic interstitial nephritis.

4. Pericarditis.

This affection being practically always a secondary affection, it necessary for its diagnosis that we obtain a history of one of the conditi giving rise to it. In the majority of cases this will be found to be ac inflammatory rheumatism, the manifestations of which are usually prominent as to make it hardly possible for them to escape detection.

Exceptions to this latter statement are to be found in those r instances in which the pericarditis itself is the first clinical evidence of t existence of the rheumatic poison. The various infectious diseases, though less frequently observed as the cause of acute pericarditis, are nevertheless important etiological factors. In the order of their frequency of action, they are tuberculosis, pneumonic fever, typhoid fever, variola, scarlatina, septicæmia, and pyæmia. Scurvy, traumatism, Bright's disease, hæmophilia, leucocythæmia, diabetes, and cirrhosis of the liver are occasional causes.

The clinical picture of acute pericarditis is a most varied one. There are instances in which the disease runs its entire course without a symptom, unless we except the physical signs, which escape attention because the rational symptoms are such as not to suggest the presence of cardiac difficulty, and therefore lead to a failure to make proper physical examinations. Indeed, it may be stated that the majority of cases of the disorder are detected only because the physician, knowing that the patient suffering from a malady capable of producing cardiac disease, makes daily systematic investigation of the action of the heart. Of the subjective symptoms, pain is the most prominent. It is not apt to be severe, and may frequently be mistaken for myalgia or intercostal neuralgia. Palpitation is often present. It is usually of mild character, but it may be so prominent as to amount to an actual delirium cordis. In the latter case it is found only in well-advanced cases, in which there is probably co-existent disease of the cardiac structure itself. Sometimes there is dyspnœa. Fever is not liable to be a prominent feature. Occasionally the laryngeal nerves are involved, in which case aphonia may be associated. The important physical signs include the pericardial friction-murmur in the initial stages, and the alterations in the area of percussion-dullness later, when effusion has taken place. For a more complete discussion of these symptoms the reader is referred to preceding pages in this chapter.

5. Endocarditis.

We recognize two varieties of endocarditis, namely: (a) Acute simple endocarditis, and (b) Malignant, ulcerative, or septic endocarditis.

(*a*) **Acute Simple Endocarditis.**—This, in the vast majority of cases, is rheumatic in origin, occurring as one of the phenomena of acute articular rheumatism in about 25 per cent. of the cases of that disease. It seems to be especially liable to attack children, the percentage of endocarditis in the rheumatic fever at that period of life being estimated as high as 65 to 80 per cent., by different authorities. The intensity of the primary disorder seems to bear no relation whatever to the incidence of the endocarditis, although Gibson has claimed that the severe cases of rheumatic fever are especially liable to be associated with endocarditis of a high grade of severity. Rheumatic fever being such an important cause of acute endocarditis has led to the ignoring of other agencies which may produce lesions. The symptoms of septic endocarditis differ from the simple variety mainly in the presence of the profound constitutional disturbance and the greater severity of the local phenomena, which are usually dependent upon involvement of almost the entire myocardium. The classification of types of septic endocarditis into typhoid, pyæmic, cardiac and cerebral, is based upon the general character of the clinical phenomena. The *typhoid types* of cases are not infrequently mistaken for typhoid fever, from which they may be distinguished by the repeated chills, the irregularity of the fever, the absence of the Widal reaction, and the presence of physical signs indicating serious cardiac derangement.

The pyæmic cases are associated with all the characteristic phenomena of pyæmia,—chills, fever and sweat succeeding each other with the irregularity belonging to that disease. Associated with these are the physical signs of endo- and myocarditis.

The *cardiac cases* give a history of long-standing valvular disease. The advent of septic endocarditis is marked by evidences of renewed cardiac disturbance, associated with the irregular fever characteristic of septic and pyæmic processes.

In the *cerebral cases* the symptoms relate almost exclusively to the brain, and include headache, delirium, and other phenomena suggestive of meningitis.

The clinical course of septic endocarditis is remarkable for the frequency with which serious symptoms, due, for the most part, to embolism, appear. These vary, of course, according to the artery obstructed. Should one of the cerebral vessels be affected, paralysis or convulsions appear; in case the pulmonary vessels are favored, respiration is suddenly embarrassed. To enumerate all the symptoms possible as secondary to embolism in this disease is a manifest impossibility.

Enlargement of the spleen and nephritis are practically always present in malignant endocarditis.

The prognosis is absolutely unfavorable.

CHAPTER XIV.

THE URINE.

NOTWITHSTANDING the valuable information to be derived from (cal and microscopical examination of the urine, the author is convince these examinations are only exceptionally made to the best advantage deficiency lies in the fact that physicians limit themselves to searchi the presence of pathological products, ignoring deficiency or excess (stituents present in all normal urines. In other words, the functior tivity of the kidneys is ignored in too many urinary examinations.

Of the functions of the kidneys we know positively that they p one that is excretory. Of late, certain data obtained from laborato periment, confirmed to a certain extent by clinical observations, ir that the kidneys are also organs of internal secretion.

Acting as excretory organs, it is the function of the kidneys to nate a standard quantity of urine, holding in solution a standard qu of excrementitious materials. Under the influence of diet, exercise perature, etc., the chemical processes of the kidneys lead to modific or variations from the standard quantities, but in health these are within circumscribed limits. Failure to appreciate these facts is a fr cause of inability to discover the presence of kidney disease.

Again, it may be stated that urinary examinations are not con with a thorough idea as to the information obtainable. Through th may obtain positive evidence of actual disease of the kidneys them positive information confirming suspicions as to disease of other c and negative information leading us to exclude certain conditions ei the kidneys themselves or of other viscera.

1. **Daily Quantity of Urine.**—The normal daily quantity o is fifty fluid ounces, or 1500 cubic centimetres. The normal daily qu for the new-born infant ranges from eight to twelve ounces. The stat to the effect that the daily quantity varies normally according to 1 ality has been made. This is undoubtedly true if the habits of a pe regards physical activity and diet are considered. The authority f statement* places the average daily quantity in the United States as ounces in men and but thirty-three ounces in women. The freest tion usually takes places in the afternoon; somewhat less during th

not been paid to this last-mentioned point, because in certain organic renal diseases the night's urine is nearly always greater in quantity than the day's urine.

Departures from the normal standard of quantity, when not habitual, are consistent with health. Thus a discharge of eighty ounces occurring occasionally may be taken as no evidence of ill-health. The influences which alter the daily quantity of urine include diet, drink, temperature, exercise, mental processes, and age.

Some foods are diuretic, notably those containing a large quantity of water, as watermelons and succulent fruits.

The drinking of large quantities of liquid, especially pure water, promotes a free passage of urine.

Hot weather and violent exercise, by producing profuse sweating, lessen urinary excretion by reason of the elimination of water by the skin, leaving less to be taken up by the kidneys.

Mental emotions, as grief, anger, etc., are not uncommonly associated with or followed by free diuresis.

The quantity of urine passed may be

a. Increased (Polyuria).
b. Decreased or suppressed (Oliguria).

(*a*) **Polyuria** may result pathologically from the following conditions :

i. *Diabetes insipidus or mellitus.* The quantity of urine in these conditions is always very large, in extreme cases of diabetes mellitus amounting to as much as 400 or more ounces daily.

ii. *Increased arterial tension*, whether dependent upon cardiac hypertrophy or interstitial nephritis. In the latter condition a general arterio-sclerosis is generally associated. The quantity of urine passed reaches 70 to 100 ounces. In one case following a temporary suppression it reached 280 fluid ounces.

iii. In the early stages of *lardaceous* or *waxy kidney*.

iv. During the *stage of convalescence from many acute diseases*, the increased flow being due to the tissue-changes attending the progress towards health.

v. In *dropsy* or *cases with serous accumulation*, as pleurisy with effusion, the daily quantity often goes above 100 ounces. The resorption of the accumulation and an associated increase in the arterial pressure are the main causative factors of this polyuria.

vi. In *hysterical seizures*, as a terminal symptom of the attacks.

vii. After *anger* or *emotional disturbances* of any kind.

viii. After *epileptic seizures* or in the course of some cases of brain disease, especially when the cerebellum is involved.

ix. After the taking of *food* containing large quantities of sugar.

x. After the exhibition of certain *drugs*, notably theobromin calomel, digitalis, the salts of potash, etc.

xi. *Phosphatic diabetes.*

(*b*) **Oliguria** occurs in the following conditions:

i. In various *acute diseases of the kidneys.* In acute nephriti of scarlatina, or pregnancy, or in the late stages of the dif ferent varieties of chronic nephritis, it may amount to ab solute suppression.

ii. In *chronic Bright's diseases*, though not usually until the ad vanced stages.

iii. In cases of disease attended by marked *dehydration of th tissues*, as by diarrhœa, vomiting, sweating, etc.

iv. In *neurasthenic states*, the deficient urination in these bein the result of defective tissue metabolism.

v. In *mechanical impediments to the escape of urine* from one both kidneys, as in ureteral obstruction by a calculu Such cases are not infrequently attended by hydronephr sis; and, as already stated on another page, the course the illness is characterized by periods of increased urina flow, during which the renal tumor diminishes in size.

vi. *Cirrhosis of the liver.*

vii. *Diminished arterial pressure*, as in diseases of the heart-mu cle, failing compensation in valvular disease, and pulm nary emphysema.

viii. In *febrile affections.*

2. **The Color of the Urine.**—The normal color of the urine h been described as a pale-yellow or amber. As the result of undue c centration this hue becomes intensified, and, by dilution, paler than t normal standard.

(*a*) **Pale urine** is, as a rule, a urine deficient in solids. A nota exception to this is found in the urine of diabetes mellitus, in which t specific gravity is high and the quantity of sugar in solution large. It noted clinically especially in the following conditions:

i. Diabetes mellitus and insipidus.
ii. Chronic Bright's disease.
iii. Hysteria.
iv. Anæmia.
v. Convalescence from acute diseases.
vi. After taking diuretic medicines.

(*b*) **Whitish urine** may be noted as

- i. The result of excessive quantity of *urates*, the cloudiness disappearing on the application of heat.
- ii. *Phosphates* (*earthy*), the cloudiness being present when the urine is passed. It disappears on the addition of a few drops of acid.

(*c*) **Yellowish or milky color of the urine** may be observed in

- i. Chyluria.
- ii. Pyuria.
- iii. Bacterial changes in the urine.

(*d*) **Yellowish-green or black urine.**

- i. A pale-green urine, as in some cases of *diabetes* with a great quantity of sugar.
- ii. *Jaundice*, owing to the presence of biliverdin or urobilin.
- iii. Poisoning by carbolic acid, hydrochinon, resorcin, and pyrocatechin.
- iv. Salol and the salicylic acid derivatives.
- v. Hæmoglobinuria.
- vi. In multiple melanotic sarcoma, in which the urine may become almost black, the color deepening as the urine becomes cooler.
- vii. After poisoning by arseniuretted hydrogen inhalations.

(*e*) **Dark-Yellow to Brownish-Red Color.**—This is the color of concentrated urines. It may be observed clinically

- i. After the ingestion of a full meal.
- ii. On a diet containing very little fluid.
- iii. After violent exercise.
- iv. From profuse sweating.
- v. In the course of fevers.
- vi. From dehydration of the body, as in severe diarrhœas.
- vii. In cases of renal congestion.
- viii. In the course of hepatic disorders.
- ix. In some cases of intestinal obstruction.

(*f*) **Reddish-brown urine** results from

The ingestion of rhubarb or senna.

(*g*) **Orange or deep-yellow** may be produced by

Santonine, and sometimes by licorice.

(*h*) **Reddish or smoky urine** occurs from

- i. Blood in the urine.
- ii. Creosote, in toxic cases amounting to a greenish-brown.
- iii. Hæmoglobinuria.
- iv. Scarlatina.

(*i*) **Brownish to a brownish-black urine** indicates the presence of

- i. Carbolic acid.

- ii. Melanin.
- iii. Methæmoglobin.
- iv. Pyrocatechin.

(*j*) **Blue color of the urine:**

- i. A cloudy, blue tint may be observed in cholera Asiatica anc typhus, owing to the presence of indigo.
- ii. A blue or greenish tint may result from the taking of indigo.
- iii. A distinct blue or bluish-green may result from the ingestio of methylene blue (medicinal).

3. **Odor of the Urine.**—The normal odor of the urine is characteristic, and is well known to every one. It has been described as aromatic but the term "urinous" is decidedly preferable. It may be altered as follows, as the result of disease or chemical changes :

- (*a*) **Ammoniacal** in cases of cystitis; or from standing for number of days in a warm place, especially if it contain much organic matter.
- (*b*) A **sugary** or **sweet odor** in diabetes mellitus.
- (*c*) A **violet odor** may occur from turpentine poisoning, or fro the oil of rosemary.
- (*d*) An **over-ripe apple** odor in acetonuria.
- (*e*) **Gangrenous** odor from destructive disease with formatio of sloughs in the urinary tract.
- (*f*) **Fæcal** odor from fistulous communication between the u nary and intestinal tracts.
- (*g*) **Special odors,** from the ingestion of food substances a drugs, notably from asparagus, garlic, cubebs, copaiba, a sandalwood oil.
- (*h*) **Sweet-brier** odor, later becoming putrid from cystinuria.

4. **Transparency of the Urine.**—Normally, the urine is perfect transparent when passed.

- (*a*) **After standing for several hours there appears a slig cloudiness, which settles to the bottom of the rece tacle,** which is due to mucus. It is not changed by t addition of alkalies or mineral acids. It is slightly i creased by the addition of acetic acid or alcohol.
- (*b*) **A cloudiness appearing on cooling** indicates urates.
- (*c*) **A cloudiness disappearing on the addition of acids** dicates earthy phosphates.
- (*d*) **A cloudiness when freshly passed** indicates phospha or pus.
- (*e*) **Cloudiness or milkiness** indicates chyluria.
- (*f*) **Cloudiness on standing for several days** is the result bacterial growth.

5. **Specific Gravity.**—The normal specific gravity of the urine ranges from 1016 to 1020. It is to be determined by an instrument known as the urinometer. Of all instruments of precision furnished for the physician, this is the most likely to be inaccurate, owing to carelessness of manufacturers. It is important that none should be used unless it has been compared with a standard instrument, and a certificate to that effect furnished. When using it, certain precautions are necessary. In the first place, any froth or bubbles on the surface of the urine should be removed by blotting-paper or a pipette, as they act as floats, preventing the instrument from sinking to the proper point. Then, too, care must be taken that the urinometer does not adhere to the sides of the vessel in which it is floated. To obviate this difficulty, the containing vessel is made of fluted glass, or various shapes are given to the air-containing receptacle of the instrument itself. When taking the reading, the eye should be on a level with the apparatus, and the figure opposite the lowest portion of the surface of the urine taken as the correct one. Care must be taken that sufficient urine is in the jar to float the urinometer. In other words, the instrument must be clear of the bottom of the jar. Attention should also be paid to the temperature of the urine under examination. As most instruments are standardized for the ordinary room temperature, this point is not of much importance; but when a freshly-passed specimen is being examined, the reading will be too low, unless account is taken of the temperature. The rule adopted for making allowance for temperature differences is as follows: "Add one degree of specific gravity for every seven degrees of temperature of the urine above that for which the urinometer is standardized."

It may be accepted as a rule that the specific gravity is in inverse ratio to the intensity of the color of the specimen. An important exception to this statement, however, is found in diabetes, in which disease the urine is pale, but the specific gravity high, owing to the presence of sugar.

The degree of specific gravity depends entirely upon the relative amount of solids in the urine. It is increased in all concentrated urines, and in most cases with oliguria. It is lowered in cases of interstitial nephritis owing to the deficient elimination of urinary solids, notably urea; in cases of neurasthenia owing to defective metabolism; and in hysteria.

An approximate estimate of the amount of the urinary solids as obtained from the specific gravity and the daily quantity of urine passed is a good index of the manner in which the kidneys perform their functions. Hence, if, in the course of a chronic renal disease, there is a sudden lowering of the specific gravity, the urinary quantity remaining as before, there is a strong probability of impending uræmia.

The method for estimating the amount of urinary solids is as follows: Multiply the last two figures of the specific gravity by Haeser's coefficient, *i.e.*, 2.33, and this gives the solids in 1000 c.c. of urine. Fror this result may be calculated, by the simple rule of three, the daily excretio of solids in the total quantity of urine passed.

Haines, of Chicago, suggests another method. He multiplies the las two figures of the specific gravity by the number of ounces passed, an this result, again, by 1.1, the final figure being the number of grains of solid passed daily. In health, 50 per cent. of the solids consists of urea. It i different, however, in disease. Hence the determination of the urea secr tion must be made by a special process.

6. **Reaction of the Urine.**—The normal chemical reaction of th urine is acid. It should be determined by means of litmus paper. Blu litmus paper is changed red by acid urine, and red paper blue by alkalin urine. In certain instances samples of urine are found in which the rea tion is said to be amphoteric, as they change blue litmus red, and red litm blue. This phenomenon is believed to be due to the presence of acid an neutral sodium phosphate. An accurate estimate of the actual degree acidity or alkalinity of the urine is rarely made in clinical work, as the i formation is not generally regarded as worth the additional trouble. seems to the writer, however, that such an investigation may be found be of advantage if systematically carried out. The process is not a dif cult one, especially if the procedure for determining the total acidity of t stomach-contents be remembered. Ten c.c. of urine, with a 1 per ce solution of phenolphthalein as an indicator, may be titrated with a de normal solution of sodium hydrate. Then the number of cubic cen metres of sodium hydrate solution used to neutralize the acid urine sho be multiplied by 10, and the result accepted as an indicator of the deg of urinary acidity. The alkalinity of other specimens may be determin by titrating with a decinormal solution of hydrochloric acid, one of a per cent. of dimethylamidoazobenzol being used as the indicator.

(*a*) **Increased Acidity.**—Normally, this may occur as the result o diet consisting very largely or entirely of animal food. The acidity of urine is increased after muscular exercise, and by the taking of acids a saccharin. It is interesting to note that the degree of urinary acidity, a rule, runs parallel with that of the stomach. Increased acidity is obser in diabetes, leucocythæmia, scurvy, and gout.

(*b*) **Alkalinity of the Urine.**—This may be a result of a diet c sisting almost entirely of vegetable food. It may also be produced by l of gastric juice, as by repeated and persistent vomiting, and by the adn istration of alkalies. It may also occur as one of the phenomena of debi and anæmia.

Ammoniacal alkalinity is usually the result of decomposition urine in the lower urinary tract, and is symptomatic of cystitis, uretl stricture, etc. It very rarely occurs in kidney disease.

7. **Macroscopic Appearance of Urinary Deposits.**—While certain insoluble substances in the urine present appearances to the naked eye suggestive to the experienced, the information thus gained should never be accepted as conclusive evidence without a microscopical examination.

A deposit that has been compared to **grains of Cayenne pepper,** and generally called "**brick-dust sediment,**" indicates the presence of *uric acid.* Its quantity is never large.

The term "brick-dust sediment" is also applied to the deposit of **amorphous urates,** which may be observed even in normal urines when exposed to the action of unusual cold. The appearance is best described as reddish and mealy.

An **abundant white deposit** suggests pus or phosphates. When the former, the sediment is creamy.

8. **Pneumaturia.**—Gas can only exist in the urine in the presence of certain forms of fermentation or the existence of a fistulous communication with the intestinal tract. It may positively be detected when present by passing the catheter, and withdrawing the urine while the outer end of the instrument is kept under water. As the result of fermentation changes, it occurs only in rare cases of cystitis.

Chemical Analysis of the Urine.

In the clinical investigation of disease we are called upon quite often, indeed, it should be a routine procedure, to examine the urine chemically and microscopically to determine (1) the presence of abnormal constituents, and (2) to determine the proportion in which the normal constituents are found. No abnormal constituent is searched for with such frequency, nor, I might say, is there one concerning which greater mistakes are made, than albumin, the presence of which, in the urine, we call albuminuria.

Albuminuria.—The most commonly-employed test for the detection of albumin in the urine is the **heat and acid test.** In the application of all tests, let it be remembered that *it is important to filter the urine, if it is not already absolutely clear,* as otherwise small quantities of albumin will escape detection. A small quantity of the suspected urine is boiled in a test-tube. Then a drop or so of nitric acid is added. If albumin is present, a cloudiness will appear. *Cloudiness appearing on heating may be due to phosphates; but, if so, it will disappear on the addition of the acid.* It is important that the acid be added after heating, and not before. The quantity of nitric acid added should never be more than 1 per cent. of urine boiled.

A very useful modification of the heat and acid test is as follows: A test-tube is filled two-thirds full of urine, the specific gravity of which has been raised several degrees by the addition of a saturated solution of common salt. This mixture is acidulated with a few drops of acetic acid. The upper layer is then boiled, and, if albumin is present, the appear a cloudiness which is readily perceived in contrast with th urine in the lower part of the tube. Very small quantities of albumi be detected by this method.

The **cold nitric-acid test,** known as **Heller's test,** is a hand but is not as sensitive as some others. Still, it is said that quantitie buminuria not detectable by it may be disregarded clinically. A fe timetres of cold nitric acid are poured into a test-tube. This is o with a similar quantity of urine. At the line of junction of the two there will appear a white band, if albumin is present.

Purdy's Ferrocyanide-of-Potassium Test.—To a test-tub half full of urine is added one drachm of a five per cent. solution of cyanide of potassium. Then add 10 to 15 drops of acetic acid. If al is present, a cloudiness in proportion to its quantity will appear. Tl does not precipitate mucin.

Tanret's test has the advantage of being exceedingly delica also open to the objection of reacting to other substances than al The solution is prepared as follows :

Potassium iodide,	3.32 grammes.
Mercuric chloride,	1.35 grammes.
Acetic acid,	22 c.c.
Distilled water, q. s. ad,	200 c.c.

The test is made by the contact method, as with cold nitric aci ler's test).

Platt's picric-acid test is probably the most satisfactory met employing that substance for the detection of the presence of al First, a solution of cane sugar of specific gravity of about 1060 is pr and with this a saturated solution of picric acid is made. The test made by the contact method ; if albumin is present, a white ring v pear at the line of contact of the two fluids.

The **picric-acid test with Esbach's solution** is especially for making approximate quantitative analyses of albumin. The sol prepared as follows :

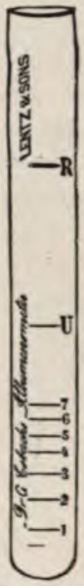
Picric acid,	10 grammes.
Citric acid,	20 grammes.
Boiling water,	1000 c.c.

For quantitative estimation of albumin, this solution is used in t bach albuminometer. The tube (see Fig. 113) is filled to the mark " U filtered urine ; to this is added the reagent to the mark at " R." ness will appear at once if albumin is present. Its quantity is dete by permitting it to settle, when it may be read off by the graduati the tube. The highest percentage, it is noticed, is .7 of one per ce the urine contains a larger proportion than this, it should be diluted before testing, and a calculation made accordingly.

Another **rough method for quantitative determination of albumin** is that of permitting the urine to settle after applying the heat and acid test, and noting the proportion the sediment bears to the whole. Thus we note one-fourth albumin, one-fifth, etc. It is incorrect to speak of the proportion of albumin obtained in this way as 25 per cent., etc.

The **albumoscope** consists of a U-shaped tube mounted on a stand. The arms are of equal length, but one is of very fine calibre and is surmounted by a small funnel; the other is of the diameter of an ordinary test-tube. Urine is first poured in the latter; acid or the test reagent into the funnel. In this way, the two fluids are brought into contact, with a sharp line of demarcation between. While intended for the application of Heller's nitric-acid test for albumin, this instrument will be found invaluable in the many instances in which one wishes to employ the contact method of testing.

FIG. 113.



Esbach's albuminometer.

The Clinical Significance of Albuminuria.—Albuminuria may occur under the following circumstances:

a. Functional albuminuria, including muscular, dietetic and cyclic albuminurias.
b. In the course of certain nervous affections.
c. In dyspepsia.
d. In the essential anæmias, including leukæmia and exophthalmic goitre.
e. Acute and chronic renal diseases.
f. Renal congestion, with heart diseases and emphysema.
g. During pregnancy and the puerperium.
h. Obstruction of the ureters.
i. Lead-poisoning.
j. In digestive disturbances.

(*a*) *Physiological Albuminuria.*—The discussion as to the possibility of the existence of an albuminuria without organic disease has been carried on with far more energy than scientific feeling. To the writer it seems inconceivable that such a condition can arise from physiological causes. Certainly an albuminuria occurring as the result of violent exercise, excessive mental labor, long immersion in cold water, or masturbation, cannot be regarded as physiological, for in each case there can be no question of the probability of an associated change in the renal circulation.

Careful examination of the history of many of these cases, and the study of their subsequent careers, will show that the temporary albuminuria is either the terminal urinary condition of a convalescing disorder or that it is the beginning of chronic renal disease. Many of the organic albumir urias are first evident only after muscular exertion, or at least in the afte part of the day, the symptom being absent in the morning. The same re mark applies with equal force to the dietetic varieties. Then, too, there still another class of cases in which a temporary albuminuria is one of th symptoms of interstitial nephritis. In each and every case of this, of all albuminurias, the symptom is to be studied in association wit other clinical phenomena before attempting to attach to it its prop significance.

(*b*) *Albuminuria in the Course of Certain Nervous Affections.*—It h been demonstrated by certain neurologists that albuminuria is a comm attendant upon many cases of functional nervous disorder; but the quanti of albumin is so small as to be detectable only by the most delicate tes Indeed, ordinary methods would fail to find it. Occasionally, however, it found in notable quantity, and is then due to metabolic abnormalities or changes in the blood.

It is not very uncommon also in organic diseases of the brain, notab in cerebral hæmorrhage and delirium tremens. In both of these affectio it is an open question whether or not the urinary condition is due wholly that of the brain. Apoplexy is associated in a very large proportion cases with interstitial nephritis and general arterio-sclerosis; hence album uria is to be expected. At the same time, it is a common experience find the quantity of albumin greatly increased immediately after the seizu The habits of delirium-tremens patients make it more than probable t there are renal changes associated; hence, while it is undoubted that all minuria may result from it, *per se*, such a position is untenable as appl to them all.

Albuminuria is rarely observed after epileptic seizures. It is m common; however, in cases of the status epilepticus, and after long-c tinued convulsions of organic brain disease. This fact must be borne mind lest the presence of albumin in large quantity leads to the diagn of uræmic convulsions.

(*c*) *Febrile Albuminuria.*—Under this term is included all those c of albuminuria occurring during the course of febrile affections. Exp ence shows that two classes of cases are liable to occur under the circ stances: (i) An *albuminuria due to a true nephritis*, best illustrated by acute nephritis of scarlatina, and (ii) *albuminurias due to the influence of fever itself and its toxin upon the renal parenchyma and its circulation.* latter only can be considered as examples of true febrile albuminuria. symptom is the most marked at the height of the fever, and disappear the temperature falls. It seems to occur with especial frequency in course of typhoid fever. In the early stages of the fever it is appare due to spasm of the arterioles, *i.e.*, anæmia of the kidneys; later, w toxæmia sets in, to that condition and to the associated lowered vascular pressure, *i.e.*, to congestion of the kidneys. The albuminuria of typhoid fever seems to bear a well-defined relation to the severity of the primary disorder, occurring early and in larger quantity in the severe cases. Its subsidence may be accepted as giving prognostic information. Thus, if it persists beyond the fifth to the eighth days of convalescence, it may be regarded (in the mild cases) as indicating a relapse. In severe cases it is not expected to disappear until after the tenth day.*

(*d*) *Albuminuria from Circulatory Disturbances.*—This class includes the albuminurias associated with valvular disease of the heart with ruptured compensation, dilatation of the heart, fatty degeneration of the heart-muscle, and pulmonary emphysema. The quantity of albumin may be quite large. Physical examination will discover associated enlargement of the liver. The pressure of abdominal tumors may so modify the renal circulation as to produce albuminuria.

(*e*) *Albuminuria in the Essential Anæmias.*—It is only exceptionally that one meets with an albuminuria even in well-advanced cases of anæmia. The best example in my personal experience occurred in a young girl with chlorosis, and in whom the albumin was in large amount, but was noticed only in the evenings' urines.

(*f*) *The Albuminuria of Acute and Chronic Renal Disease.*—It is in this connection that albuminuria has been most studied. Indeed, for many years albuminuria was regarded as pathognomonic of Bright's disease. The fact is that albuminuria may exist with sound kidneys, and may be absent when these organs are extensively degenerated.

In *acute parenchymatous nephritis* the albuminuria is prominent, and is a constant symptom. The quantity of urine being small, the actual loss of albumin to the system is relatively small, rarely over 20 grammes daily.

It is also large in amount and constant in *chronic parenchymatous nephritis*. Quantitative analysis may show it to be as high as 1.5 to 3 per cent. The daily loss to the system may amount to from 15 to 30 grammes.

It is in connection with *chronic interstitial nephritis* that albuminuria is most misunderstood, for in this disease it is both small in quantity and irregular in its presence. The small percentage is well shown by the fact that, notwithstanding the polyuria so commonly present, the total daily loss of albumin does not exceed 2 grammes in the majority of cases, and is rarely over 5 grammes. Hence it can never be a cause of the anæmia in cases of this disease. On the other hand, albumin is so frequently absent from the urine of interstitial nephritis that we cannot regard it as a diagnostic symptom, nor its absence as possessed of much special clinical significance.

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* See Simon's quotations from Robin, CLINICAL DIAGNOSIS, p. 377.

In *amyloid degeneration of the kidneys* we find remarkable vari in the daily quantity of albumin. A point worthy of especial note i diagnosis of this disease is that the albumin consists of a larger prop of serum globulin to serum albumin than in any other condition.

(*g*) *Albuminuria of pregnancy and lying-in.* The best clinician assert that the albuminuria of pregnancy is due to a nephritis. Thei remain those who claim that it is dependent upon toxic conditions blood brought about by the pregnancy, and others who attribute it t turbances in the renal circulation by the pressure of the enlarged u and still others to an impeded outflow of urine through the ureters.

(*h*) *Obstruction of the ureters.*

(*i*) *Lead-poisoning.* The majority of cases of albuminuria depe upon lead-poisoning are the results of interstitial nephritis.

(*j*) *Digestive disturbances.* Albuminuria from this cause gen occurs when the diet has been rich in nitrogen, and especially when raw. It is brought about through the medium of reflex, nervous, motor or trophic changes affecting the kidneys.

In all cases of albuminuria it is important to determine wheth albumin discovered is due to

(i) A true albuminuria, or
(ii) Accidental albuminuria, in which the albumin is due t
mixture of an albuminous fluid like pus or blood.

In accidental albuminuria the albuminous substance, *i.e.*, pus or l may be derived from the urinary tract or from the vagina (leucor menstrual blood, serum) in women, or pus, serum and blood froı male urethra.

Globulinuria or Serum Globulin in the Urine.—Gl is precipitated in all the albumin tests, but constitutes such a rela small proportion of the precipitate that it may safely be disregarc most instances.

While serum globulin is soluble in a saline solution like the ur is insoluble in pure water. Hence, Roberts has suggested as a test presence that a drop of the suspected urine be dropped into distilled when, if globulin is present, a cloudiness follows the descent of the After the addition of a large quantity of the urine, the mixture be quite cloudy; but this opacity is dissipated by the addition of a d acetic acid.

Another test is as follows: Render the urine alkaline by the ad of liquor potassæ. Then underlie the specimen with a saturated so of magnesium sulphate. If globulin is present, a well-defined rin appear at the junction of the two fluids.

Globulin never occurs in the urine excepting in association with albumin. Hence its significance is that of albumin. A special poi its discovery is in cases of suspected amyloid degeneration of the kidneys, in which disease globulin is present in greater proportion to albumin than in other conditions.

Peptonuria or Peptones in the Urine.—Even a casual perusal of medical literature on this subject shows that authors hold widely-diverse views concerning it. Inasmuch as they are all clinicians noted for accuracy of observations, it is a fair presumption that they must be at variance concerning what constitutes peptonuria. Accepting Kühne's definition of "peptone," a peptonuria cannot exist, as that substance is not found elsewhere than in the contents of the stomach. Accepting the older definition of peptone, peptonuria is a common clinical phenomenon. Such a peptonuria is in reality an albumosuria; but inasmuch as the albumose in question is chemically distinct from others, and presents a clinical significance peculiarly its own, and as the clinical study of disease alone concerns us, we may with this understanding continue the use of the term "peptonuria."

Von Jaksch is the great authority for the importance of peptonuria as a symptom. He says that its presence is "associated with such processes as are characterized by the collection and subsequent destruction of leucocytes under such circumstances that the products of disintegration, including the peptone constituent of these bodies, can obtain entrance into the blood stream, to be subsequently eliminated by the kidneys." Peptonuria arising in this way is known as "pyogenic peptonuria." It occurs in the course of suppuration, if conditions favor the absorption of the pus, in the stage of resolution of croupous pneumonia, empyema, etc. Before one assumes, however, that a peptonuria is indicative of suppuration, he must first exclude the other known causes of the condition.

Thus peptonuria may be histogenic or hæmatogenic when it occurs in the course of scurvy, phosphorus poisoning, pregnancy, and psychoses. It is a normal condition of the puerperal period. During pregnancy, its prominence is suggestive of death of the fœtus.

An enterogenic peptonuria occurs in the course of intestinal ulcerations, as from typhoid fever, intestinal tuberculosis, and intestinal carcinoma. Such a peptonuria is due to the direct passage into the blood, through the ulcerated parts, of the peptones derived from the food into the blood.

The Determination of Peptonuria.—The majority of methods for the clinical recognition of peptonuria are too complicated for the demands of medical practice. Salkowski's method is, however, readily applied, and gives results as accurate as those technically exact. It has received the approbation of von Jaksch and Simon. It is as follows:

"Fifty c.c. of urine are acidified in a beaker with 5 c.c. of hydrochloric acid, and precipitated with phosphotungstic acid, the mixture being heated over the free flame, when in a few minutes the precipitate will form a resinous mass which closely adheres to the bottom of the vessel. The supernatant fluid is decanted off, and the mass at the bottom, which has become granular, is washed twice with distilled water, which is likewise removed by decantation. The precipitate is then covered with about 8 c.c. of distilled water and treated with 0.5 c.c. of a sodium hydrate solution (sp. gr., 1.16). Upon shaking the beaker the mass will dissolve, the solution assuming a dark-blue color. This is heated on the free flame until the blue color turns to a dirty grayish-yellow; the solution at the same time becomes turbid, but at times may turn yellow and remain clear. This decoloration may be hastened by the further addition of a few drops of the sodium hydrate solution. As soon as this point has been reached, some of the liquid is placed in a test-tube, allowed to cool, and then treated with a very dilute solution of cupric sulphate (1 to 2 per cent.), drop by drop, when, in the presence of peptones, the solution assumes a bright-red color, which may be brought out still more strongly if the specimen is now filtered. If albumin or much mucin be present, these bodies must first be removed." (Simon.)

Mucinuria.—All urines contain a small quantity of mucus. I health, however, this quantity is so small that the essential constituent o mucus, namely, mucin, cannot be discovered by ordinary tests. An exces of mucin in the urine possesses the same significance as excess in mucus Hence it is found in all catarrhal inflammations of the urinary apparatus especially in cystitis and pyelitis. In these diseases the quantity may be s large as to form ropy or gelatinous masses in the urine. It is not neces sary to have actual inflammation, as mucinuria accompanies condition characterized by irritation of the lining membrane of the urinary apparatu This may occur in fevers, especially in pneumonia, typhoid fever, pleuris and intermittent fever.

Test.—Mucin is rendered soluble in the urine by the chloride sodium. We therefore, in testing for its presence in the urine, lessen i solubility by the addition of an equal quantity of water. An excess acetic acid is next added, and any precipitate resulting is mucin.

Albumosuria or Albumoses in the Urine.—Under this ter are included certain conditions characterized by the presence of prope tones in the urine. Attempts have been made to show that this substan is really a mixture of no less than four different proteids. However t may be, clinical knowledge of the subject is distressingly inexact, certai sufficiently so to make it impossible to tabulate any conclusions. Albu suria has been observed in a variety of diseases, in all of which it has li wise been found absent. Thus it has been noted in osteomalacia, der titis, and intestinal ulceration.

Determination of Albumosuria.—Albumoses precipitated by usual tests for albumin are redissolved on the application of heat. Hen if urine subjected to the salt, acetic acid and heat test for albumin is filter the albumoses will pass in solution in the hot filtrate, and will precipitate on cooling. If to the hot filtrate sufficient sodium hydrate solution to make it alkaline be added, and then a weak solution of cupric sulphate, drop by drop, a red color will result.

Boiling of the filtrate with Millon's reagent will also give a red color when the albumoses are present. *Millon's reagent* is prepared as follows: Dissolve 1 part of mercury in 2 parts of nitric acid of a specific gravity of 1.42, and dilute with 2 volumes of water.

Sugar in the Urine; Glycosuria.—The presence of sugar as a constituent of the normal urine has been argued warmly, existing views favoring the idea that it is present, but in such small quantities as to be undetectable by ordinary clinical tests. Urine containing sugar is usually of high specific gravity, *i.e.*, from 1030 to 1040, and exceptionally as high as 1050. Too much reliance, however, must not be placed upon this fact, for sugar has been discovered to the extent of 1 per cent. in urine having a specific gravity of 1007. As a rule, too, the urine is of a pale color; but here, again, reliance must not be placed upon this fact.

Sugar may appear in the urine as a temporary condition; as a symptom in a variety of diseases, notably in elderly persons shortly after breakfast; during convalescence from acute diseases; in a number of nervous diseases, as epilepsy, hysteria, sciatica, cerebro-spinal meningitis; injuries to the brain, especially in the vicinity of the floor of the fourth ventricle; obesity; intermittent fever, especially after the paroxysms; scarlatina, cholera, gout, diseases of the lungs, diseases of the liver. It may result from dietetic causes, *i.e.*, from the excessive ingestion of foods containing sugar. In this connection it should be observed that the kind of sugar taken in excess is the kind that will appear in the urine. Von Noorden has demonstrated that the maximum quantities of the sugars that may be taken by healthy individuals without producing glycosuria are as follows: Grape sugar, 200 to 250 grammes; fruit sugar, 200 grammes; cane sugar, 150 to 200 grammes; milk sugar, 120 grammes. The ability of large quantities of sugar to produce glycosuria has been utilized by von Jaksch and Clifford Mitchell in the early diagnosis of diabetes. The former administers 100 grammes of cane sugar, and, if it produces glycosuria, concludes that the patient is in the early stage of diabetes. Mitchell employs champagne for the same purpose, as that beverage in small quantities is manifestly bad for the diabetic, increasing his percentage of sugar in the urine.

Occurring as a persistent symptom, glycosuria is to be regarded as diagnostic of diabetes mellitus, in which case it will be associated with emaciation, prostration, thirst, dryness of the mucous membranes, polyuria, loss of sexual appetite, pruritus, and numerous nervous manifestations.

Tests for Sugar.—Moore's or Heller's Test: Equal parts of liquor potassæ and the suspected urine are mixed in a test-tube, and the upper layer of the mixture boiled. If sugar is present, there will be a change of color ranging from a dark yellow to black, according to the quantity of sugar present. This test is not a delicate one, as it does not detect sugar in less than 0.3 per cent. The presence of albumin interferes with it, hence that substance should be removed by the heat and acid test and filtration before applying the test. It should also be remembered that all high-colored urines undergo some darkening of color by the addition of liquor potassæ and boiling.

Trommer's Test.—This test is used very largely because of the convenience of preparing the solution and its readiness of application. To a mixture of equal parts of the suspected urine and liquor potassæ a few drops of a weak solution of cupric sulphate are added. It is important that the quantity of the latter should not be too large; enough to impart its color to the mixture is sufficient. On adding the copper solution there will appear at once a precipitate of blue cuprous oxide, which is immediately dissolved if sugar is present. The mixture is now heated, and the sugar causes a further oxidation of the copper, shown by the appearance of a reddish color, and ultimately of a yellowish-red precipitate of cuprou oxide. The boiling should not be prolonged, otherwise the copper will b precipitated by other substances in the urine. This test is capable of detecting the presence of 0.5 to 1 per cent. of sugar in the urine.

Fehling's Solution.—The test-solution is prepared as follows:

Cupric sulphate, ...	34.64 grammes.
Sodio-potassium tartrate, ...	173 grammes.
Caustic soda solution (sp. gr. 1.12), . . .	480 c.c.
Water, q. s. ad, ...	1000 c.c.

Each cubic centimetre of this solution is equivalent to 0.005 grm. of suga A portion of this test-solution is boiled in a test-tube. If the copper not reduced in the act, the preparation is fit for use. Then the suspecte urine is added, drop by drop, until the characteristic reaction takes plac but the quantity added must never exceed that of the test-fluid. It important to avoid prolonged boiling after the addition of the urine, other substances may thus reduce the copper.

Fehling's solution does not keep well; hence it has been recommended that the sodio-potassium tartrate and caustic soda be dissolved 500 c.c. of water, and the cupric sulphate in another 500 c.c. of wat Each of these solutions is to be kept in a well-corked bottle. For u equal parts are mixed to make Fehling's solution. Prepared in this w the test liquids will keep indefinitely.

Phenyl-Hydrazin Test.—To about a fluid drachm of urine in a te tube, about 5 grains each of phenyl-hydrazin hydrochlorate and sodi acetate are added. The tube, with its contents, is then placed in a wat bath with boiling water, and kept boiling for from twenty minutes to half an hour. It is then placed in cool water, when, if sugar is present, yellow crystals of phenyl-glucosazone will be precipitated. If sugar is present in abundance, they will be perceptible to the naked eye. If the quantity of sugar is very small, a portion of the sediment may be removed by means of a pipette, and examined under the microscope, when the crystals will appear, needle-shaped crystals forming small spherules; or, again, they may be isolated, or in small groups. This test is exceedingly delicate, as it is capable of detecting one-fortieth of a grain of grape sugar to the ounce of urine. The test, though somewhat tedious, is an invaluable one, as it is not produced by any other substance in the urine than glucose.

Haines's Test.—This is an exceedingly handy test, the test-solution being perfectly stable. It is prepared as follows:

Pure cupric sulphate, ...	30 grains.
Distilled water, ...	½ fl. oz.

Make a perfect solution, and add

Pure glycerin, ...	½ oz.

Mix thoroughly, and add

Liquor potassæ, ...	5 fl. oz.

To use this fluid, take about a drachm of it and boil it in a test-tube. Then add the suspected urine drop by drop, until the reaction of sugar is obtained; but do not add more than eight drops.

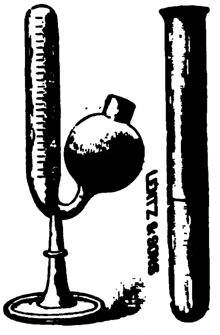
The substances in the urine which interfere with the reliability of the copper tests, by reason of their ability to oxidize the cupric sulphate, are uric acid, creatinin, coloring matters, and bile-pigments. In cases in which the copper reaction is doubtful, it is wise to employ the fermentation or phenyl-hydrazin test. Or the urine may be filtered through animal charcoal until entirely colorless, when the selected copper test may be employed.

Picric-Acid Test.—Equal parts of the suspected urine and saturated solution of picric acid are mixed in a test-tube, and half a fluid drachm of liquor potassæ added. An orange-red color results. If, now, the contents of the tube are heated, they will change to a dark mahogany color if sugar is present.

Quantitative Testing for Sugar. The Fermentation Test.—Sir Wm. Roberts has proposed a very practical method of estimating the percentage of sugar in the urine by determining the differential densities of the urine before and after removal of the sugar by fermentation. Four ounces of the urine are placed in a bottle along with a small piece of compressed yeast. The cork of the bottle is nicked, to permit of the escape of the carbonic acid gas evolved in the fermentation which ensues. Another bottle, containing the urine, but without yeast, and well corked, is also prepared. Both bottles are placed in a warm place for from twelve to twenty-four hours. The specific gravities of the two specimens are then taken, and the difference noted. For each degree of specific gravity lost, one grain to the fluid ounce of urine is estimated. If this figure be multiplied by 0.23, we obtain the percentage quantity of sugar. The only objection to be urged against this test is the time required.

Einhorn has devised a little apparatus which he calls a *saccharimeter*, and which is useful for estimating the quantity of sugar in diabetic urine. It is a U-shaped tube with a bulb on the short end, similar in construction to the Doremus ureometer. The long closed arm is graduated in fractions of a gramme, by means of which the percentage of sugar, as indicated by the extent of the fermentation from yeast, may be read off. Urines containing large quantities of sugar should be suitably diluted before employing this instrument.

FIG. 114.



Einhorn's saccharimeter.

Quantitative Estimation of Sugar by Fehling's Solution.—Ten c.c. of urine are diluted with water to 100 c.c. and placed in a burette graduate in cubic centimeters. Ten c.c. of the test-solution ar diluted with an equal quantity of water and placed in flask, which is set on a wire-gauze support on a retor stand and boiled over an alcohol flame. While th solution is boiling, the urine is added from the burette drop by drop. The process is continued until all th copper in the test-fluid has been reduced, when th quantity of urine used may be noted. This quantit is equivalent to 0.05 of sugar, and from this may be calculated the quantit of sugar in the daily urinary excretion, the latter figure being know Thus, if 45 c.c. of the mixture of urine and water have been used to reduc the copper in 10 c.c. of Fehling's solution, 4.5 c.c. of urine contain 0.0 gramme of sugar. If the daily quantity of urine is 2000 c.c., then the dail excretion of sugar must amount to 22.22 grammes.

Purdy's Method.—The standard solution used by Purdy for th quantitative determination of sugar in the urine is as follows :

Cupric sulphate, ...	4.742 grammes.
Potassium hydrate, ...	23.30 grammes.
Strong ammonia (sp. gr., 0.9), ...	450 c.c.
Glycerin, ...	38 c.c.
Distilled water, q. s. ad, ...	1000 c.c.

The cupric sulphate and glycerin are dissolved in 200 c.c. of wate and the potassium hydrate in another 200 c.c. of water. The solutions a then mixed, and when cold, the ammonia is added. Then distilled wat is added until the whole equals 1000 c.c. Thirty-five c.c. of this solution represent 0.02 gramme of glucose.

The test is then proceeded with in a way similar to that used in the case of Fehling's method. Thirty-five c.c. of the test-solution are mixed with two volumes of water, placed in a flask, and boiled over a spirit lamp. A burette is filled with the suspected urine, and its contents emptied, drop by drop, into the boiling liquid, until the blue color of the same is discharged completely. The quantity of urine used is then noted, and this represents exactly 0.02 gramme of sugar. From this may be calculated the quantity of sugar in the 24 hours' urine.

Acetone; Acetonuria.—Acetone is found as a normal constituent of the urine, but in very small quantity, *i.e.*, about 0.01 gramme in the 24 hours. Its quantity may be increased physiologically by an exclusively albuminous diet, persisted in for several days. Pathologically, it is found in a number of diseased states characterized by increased albuminous decomposition. These include the following:

1. **Febrile states,** in which the duration of the pyrexia seems to be a more important factor than its intensity, for acetonuria has been observed in typhoid fever, scarlatina, croupous pneumonia, acute miliary tuberculosis, acute articular rheumatism, and septicæmia; while it has been absent invariably in fevers of short duration.
2. **Diabetic acetonuria.** This is really the most important variety of acetonuria, the prominence of the symptoms being in direct proportion to the intensity of the primary disease.
3. Acetonuria accompanying **cancer.** This may be called a cachectic acetonuria. It seems to bear a relation to the cancerous process, as it occurs independently of the starvation which one observes in many cases late in their course.
4. Acetonuria of **starvation.**
5. **Psychotic** acetonuria.
6. Acetonuria as an expression of **auto-intoxication.**
7. Acetonuria in **derangements of digestion.**
8. Acetonuria in **chloroform narcosis.**

Tests for Acetonuria. Ralfe's Test.—This is probably the readiest of application. Twenty grains of iodide of potassium are to be dissolved in one drachm of liquor potassæ, and boiled. Then the test is made by the contact method, the urine being poured carefully on the solution. At the line of junction of the two liquids there will appear a white cloud of precipitated phosphates. If acetone is present, this will gradually give place to a yellow color from the formation of iodoform crystals. This test is a comparatively rough one. It may be made more delicate by first distilling the specimen of urine, and using the distillate for the examination.

Chautard's Test.—If a drop of an aqueous solution of magenta which has been decolorized with sulphuric acid is added to urine containing more than 0.01 per cent. of acetone, a violet color will appear. When acetone is present in minute quantities, it may take five minutes for the reaction to appear.

Lieben's Test.—Add a little phosphoric acid to about half a litre of the urine, and distill. To a small quantity of the distillate add a few drops of iodo-potassic-iodide solution and caustic potash. Acetone will cause a precipitate of yellow iodoform crystals, which will be recognized by their odor. Von Jaksch is the authority for the delicacy and reliability of this test.

Diacetic Acid; Diaceturia.—Diacetic acid as a constituent of the urine is always pathological. It possesses about the same significance as acetonuria, being a prominent feature of diabetes and fevers. Its presence in diabetes may be regarded as unfavorable, as it is present only in bad cases, and absent in the mild ones. Its occurrence in the febrile affections of children is not unusual, and not to be regarded as necessarily unfavorable. In adults, however, its advent is always a grave sign, as it is then usually followed by coma within a short time. It seems to be especially common in cases of diabetes in young subjects.

There is a form of diacetæmia which appears to be an idiopathic affection, and which is characterized by vomiting, dyspnœa, jactitations and diaceturia, followed by coma and death.

The **best test for diacetic acid** in the urine is that of von Jaksch. "To the urine a fairly concentrated solution of perchloride of iron is cautiously added, and if a phosphatic precipitate forms, this is removed by filtration and more of the perchloride of iron solution supplied. If the Bordeaux-red color appears, one portion of the urine is boiled, whilst another is treated with sulphuric acid and extracted with ether. If, now, the portion of urine which has been boiled shows little or no change, whilst the perchloride of iron reaction in the ethereal extract is no longer evident after 24 to 48 hours, and if, at the same time (on testing the urine, directly and its distillate), it is found rich in acetone, the condition may be inferre to be that of diaceturia."

Pus in the Urine; Pyuria.—Urine containing pus presents to th naked eye a cloudy appearance when the pus is in small quantity, or, whe it is large, a yellowish or milky color. It may be confounded with th presence of phosphates or amorphous urates. A distinction, however, i readily made, as phosphatic urine clears up on the addition of an aci while that containing pus has its cloudiness increased, or at least un affected ; from urates by the addition of heat, which clears up the urat and increases the opacity of pyuria by reason of the associated albumin ria. One should not, however, depend upon the recognition of pho phaturia or lithuria for excluding pyuria, but should apply tests for the recognition of pus. These are

a. Chemical, and
b. Microscopical.

The chemical tests include those of Vitali and Donne. **Vitali's test** is as follows: The suspected urine is passed through filter paper. A few drops of an old tincture of guaiacum, which has been kept in the dark, are then thrown on the sediment remaining in the funnel, and, if pus is present, a characteristic blue color will appear.

Donne's test is performed by adding a small piece of caustic soda to the suspected urine. In the presence of pus there will appear a thick gelatinous deposit, which adheres to the bottom of the tube.

The **iodo-potassium-iodide** test may be said to be both chemical and microscopic. Under certain circumstances it may be difficult to differentiate leucocytes from small round epithelial cells resembling them. Then the iodo-potassium-iodide solution may be added to the specimen. This stains the leucocytes a deep mahogany-brown, while the epithelial cells take on a light-yellow color.

Under the microscope, leucocytes appear as pale, finely granular spherical cells, containing usually a single nucleus, often, however, two or three nuclei. The application of acetic acid causes them to swell, sometimes to double their size, and at the same time rendering their nuclei more distinct and dissipating their granular matter. Mixture with a strong alkali, as ammonia or liquor potassæ, causes them to swell and coalesce and form a thick glairy mass,—upon this fact depending Donne's test for pus as above given. These changes are noted in strongly-alkaline urine, as in cases of cystitis; hence pus-cells cannot be discovered microscopically under such circumstances, and recourse must be had to the chemical tests.

Inasmuch as pus is an albuminous fluid, pyuria must always be associated with some albuminuria. It is important always to determine whether the albuminuria is of greater degree than the severity of the pyuria would warrant. Hottinger has demonstrated that a pyuria presenting 100,000 pus-cells per cubic centimetre of urine will produce 1 per cent. of albumin in the urine. With this information, and the counting of the pus-cells by the hæmatocytometer, one may readily detect the significance of the associated albuminuria.

The number of pus-cells in pyuria may range all the way from 1400 per cubic centimetre in mild to 150,000 per cubic centimetre in the severe cases. Hottinger has stated that the daily discharge may reach all the way from 40,000,000 to 150,000,000 pus-cells.

The presence of pus in the urine must indicate a pus-forming lesion either in the urinary tract or in some of the structures communicating with it. Hence **pyuria may originate in**

a. The urethra:
    (i) pendulous;
    (ii) prostatic;
    (iii) urethral appendages.
b. The bladder.
c. The ureters.
d. The kidneys.
e. Suppurating cavities having fistulous communication with the urinary tract.

It is important in every case of pyuria to determine the portion of the urinary tract from which the pus is derived. This may usually be done without difficulty by certain clinical, chemical and microscopical tests.

The chemical reaction of the urine offers a rough guide, inasmuch as purulent urine of acid reaction indicates a renal source, while alkaline reaction indicates vesical origin.

In **renal pyuria,** especially when dependent upon pyelitis or pyelonephritis, the quantity of pus is sometimes large, thick, and creamy separating readily as sediment, the urine not undergoing decompositior for days.

Contrasted with this picture is that of the thick, glairy pus of **vesica origin.**

An **intermittent pyuria** suggests an intermittent cause, which i usually found in renal suppuration with urethral obstruction, which is re lieved from time to time. It may also result from extra-urinary suppura tion, the fistulous communication with which becomes closed from time t time. In these cases the constitutional symptoms of suppuration affor diagnostic aid. With the damming up of the pus by closure of the oper ing appears fever, etc.; with renewal of the outlet, the constitutional symp toms subside.

The **appearance of flakes of pus** in the urine generally indicat an urethral source. It is said that those threads which are thin and lor come from the anterior urethra, and those which are broad and thick fro behind the compressor urethræ muscle.

The **three or four glass test** affords a valuable guide for the d tection of the source of the pus. First the urethra is washed out with weak solution of boric acid. If the washings contain pus-cells, it is e dent that some of the pus, at least, comes from the urethra. A portion the urine is then passed into a second glass. If this is free of pus-cells, proves that the pyuria must be urethral. If, however, it contains pus, th the pyuria must be either vesical or renal. If, now, the finger be insert into the rectum and the prostate well massaged forwards, the contents the ducts of this gland and the seminal vesicles will be forced into t urethra, whence they may be washed into a third glass by another uri tion. The finding of pus in discharges demonstrates an origin of pus in the accessory glands of the deep urethra. Vesical and renal origin may be differentiated by **Sir Henry Thompson's method.** The bladder is emptied thoroughly by a soft rubber catheter, the patient being in a standing position. It is then washed out thoroughly. The urine is then permitted to escape by the catheter into a fourth glass. Such urine must come from the kidneys direct, because, according to Thompson, "the bladder ceases for a time to be a reservoir; it does not expand, but is contracted around the catheter, and the urine percolates from the ureters direct." If this procedure determines a renal origin, we may adopt another and very simple device for determining whether one or both kidneys are diseased, and if but one, which one.

The cystoscope, demanding a high degree of technical skill for its successful manipulation, is not available for the general practitioner, although it is capable of giving the necessary information. In place of using it, we may insert a rod-like instrument into the rectum, and by it raise the floor of the bladder, forming a ridge between the ureteral orifices. In this way we have two water-sheds, one supplied by the right kidney, the other by the left. Skillful use of a beaked catheter will enable us to withdraw urine from each of these at will, examination of which will determine the source of the pus.

The **renal diseases producing pyuria** are as follows:

a. Acute and chronic pyelitis.
b. Primary and secondary pyelitis.
c. Pyelonephritis.

Primary pyelitis originates in the kidneys, and may be produced by drugs or poisons, notably turpentine, cantharides, and various balsams. It may also arise from bacterial irritation, notably after or during the course of typhus and typhoid fevers, tuberculosis, and influenza. Some cases are traumatic, resulting from a direct injury, as a blow, or from the mechanical action of a renal calculus, the history of the case being the main guide for differentiation.

The quantity of pus in pyelitis may vary considerably. The urine is always acid in reaction, and under the microscope is found to contain epithelial elements of the renal pelvis. A similar condition of affairs may occur in acute or chronic nephritis, but in these diseases the number of pus-cells is by no means as large as in pyelitis.

Associated disease of the renal substance constitutes the case one of **pyelonephritis.**

Secondary pyelitis is always an ascending inflammation starting in the bladder. It originates usually in a cystitis due to instrumentation in a gouty subject, to the sudden withdrawal of urine in prostatic retention, or to tumor or stone in the bladder. Secondary pyelonephritis is one of the common attendants upon severe cases of acute and chronic myelitis, and is a very serious complication, both as regards the life and the comfort of the patient.

In **acute parenchymatous nephritis** pus-cells are always found in considerable number, though by no means in as large quantity as the quantity of albumin would lead us to expect. More than this, we have the associated symptoms as described by the patient, and the microscopic finding.

Chronic diffuse nephritis also presents a number of pus-cells to the microscope; but they are not numerous, and we have the well-marked albuminuria, the diminished excretory function of the kidneys, and the presence of tube-casts.

Vesical pyuria, as already stated, is attended with an alkaline reaction of the urine, which is usually found to have undergone ammoniacal decomposition. Microscopic examination discovers the triple phosphates in abundance. Pus-cells are not, as a rule, discoverable, because the pus has been converted into a gelatinous, glairy mass. That pus is present is to be determined by one of the tests already given.

The **strongylus gigas** in the pelvis of the kidney may produce both pyuria and hæmaturia.

The diagnosis of **pyuria originating in an extra-urinary source** depends first upon the fact that the pus has appeared suddenly in a previously clear urine, and the history of the case, which suggests the situation of the original disease.

Blood in the Urine; Hæmaturia.—Urine containing blood presents various appearances to the naked eye according to the quantity of blood present and its intimacy of mixture with that fluid. Thus the urine may be bright-red in color to dark-brown, or even black or smoky. On standing it throws down a sediment corresponding in a general way to the color of the urine, and found on examination to contain coagula. In exceptional cases the blood is present in quantity too small to cause changes perceptible to the naked eye, in which case we must have recourse to the microscope for its detection.

The microscopic detection of blood is usually an easy matter, especially in cases in which it has been derived from the bladder, for in vesica hæmaturia the blood-corpuscles are but little changed. Sometimes they are somewhat swollen or shrunken or cremated. In renal hæmaturia however, they may undergo changes by reason of the prolonged action of the urinary water, and present an appearance of pale rings with double contour, containing no nucleus, and generally known as "blood shadows."

The chemical tests for the presence of blood tell us only that th blood-coloring matter is present. They are as follows:

(*a*) **Almen's Guaiacum-Turpentine Test.**—To 10 cubic centimetres of the suspected urine is added a mixture of tincture of guaiacum and old oil of turpentine. At the point of junction of the two liquids there will appear a white line, changing to blue if blood-coloring matters are present.

(*b*) **Heller's Test.**—A portion of the suspected urine is treated with liquor potassæ and boiled. The basic phosphates are precipitated, and carry with them the hæmatin and blood-coloring matter. The precipitate will thus present a bright-red color. If, however, there is an abundance of other urinary coloring matters, as urobilin, interfering with the satisfaction of this test, filter out the sediment. Then dissolve it by pouring acetic acid on the filter. The resulting solution will be of a red color, gradually disappearing on exposure to the air, if blood is present.

Hæmaturia may result from bleeding from any portion of the urinary tract. Its causes may be tabulated as follows:

1. **Kidneys.**
    - a. General diseases.
        - i. Scurvy.
        - ii. Hæmophilia.
        - iii. Leucocythæmia.
        - iv. Purpura hæmorrhagica.
        - v. Infectious diseases.
            - *i.* Yellow fever.
            - *ii.* Pernicious malarial fevers.
            - *iii.* Relapsing fever.
            - *iv.* Typhoid and typhus fevers.
    - b. Active congestion of the kidneys.
    - c. Acute nephritis.
    - d. Malignant diseases of the kidneys.
    - e. Embolism of the kidneys.
    - f. Chronic nephritis (interstitial or parenchymatous).
    - g. Tuberculous disease.
2. **Ureters.**
    - a. Calculus.
    - b. Uric acid.
3. **Bladder.**
    - a. Diphtheritic cystitis.
    - b. Malignant ulceration.
    - c. Stone in the bladder.
    - d. Traumatism.
    - e. Parasites.
    - f. Varicose veins.
    - g. Villous tumors.
    - h. Tuberculosis.

4. **Urethra.**
    - a. Prostatitis.
    - b. Mechanical injury (foreign body or instrumentation).
    - c. Urethritis.
5. **Extra-Urinary.**
    - a. Contamination with menstrual blood.

The differentiation of these various conditions is to be based upon a study of the history of the case, and an analysis of the symptoms present, and a microscopic and chemical examination of the urine.

It is important in any case of hæmaturia that the source whence the blood is derived be recognized. This may be done readily by paying attention to the appearances of the urine, the character of the clots if present, and the associated clinical symptoms.

The brighter and the more arterial the color of the bloody urine, the nearer is the source of the hæmorrhage to the urinary meatus. An exception to this statement is found in cases of severe injury to the kidneys in which the hæmorrhage is profuse, and in some cases of malignant disease of the kidneys. Neither of these conditions really invalidate the value of the proposition, as the attendant clinical phenomena and the history of the case are sufficient to make a diagnosis.

Blood appearing at the meatus urinarius **without a simultaneou escape of urine** must come from the urethra.

Hæmaturia with smoky color of the urine is practically alway of renal origin. The only exception to this statement is found in certai rare cases of vesical hæmorrhage in which the blood escapes slowly.

In **hæmaturia of vesical origin** the blood-cells present a norma appearance, unless the urine has undergone ammoniacal fermentation, i which case numerous "blood-shadows" may be seen. The blood is not a intimately mixed with the urine as in renal hæmorrhage, and subsides rap idly in a conical glass. The blood-clots are, as a rule, large and irregula

Hæmaturia appearing after slight indirect injury is strongl suggestive of calculus or malignant disease. If not associated with oth symptoms, it is probably due to calculus or to chronic Bright's disease. I the case of malignant disease of the bladder or kidneys, there will be foun pain and other symptoms to suggest the diagnosis. If the hæmorrha disappears on rest, stone is to be suspected. Benign growths are to be su pected when the history of the bleeding extends over a number of yea The hæmorrhage in these cases may be profuse ; sufficiently so, at times, endanger life.

The relation in point of time in the act of micturition affords some i formation. Hæmorrhage appearing at or near the close of urination su gests a vesical or prostatic source.

The appearance in the urine of **long earthworm-like clots** tells that the blood has passed through the ureters, and that the hæmorrhage must be either renal or ureteral,—usually, however, the former.

Renal hæmaturia is suggested by the intimate mixture of the blood with the urine, by the smoky color of the mixture, by the relatively small quantity of blood, and the presence of a large percentage of albumin, tube-casts, and a diminished urea secretion, and other symptoms suggestive of renal disease.

The various forms of Bright's disease constitute the most frequent cause of hæmaturia. The symptom is most frequently observed in connection with acute parenchymatous nephritis. The quantity of blood lost is usually small.

In **hæmorrhage from the pelvis of a kidney,** the lesion is usually unilateral, which may be determined by catheterization of the ureters or by the "water-shed" test, as described under pyuria. It is commonly due to renal calculus, and is apt to intermit.

Age is of some value in determining the cause of hæmaturia.

Occurring *before the age of five years*, scurvy and renal concretions are the most probable causes.

Between puberty and twenty years of age, tubercle of the bladder and renal stone are to be suspected.

Between the ages of twenty and thirty, gonorrhœal prostatitis.

Between the ages of thirty and forty, vesical papilloma.

After the age of fifty, malignant tumor and vesical stone.

It is important to bear in mind that in all cases of hæmaturia *instrumentation for diagnostic purposes is not to be employed unless absolutely necessary, and then only the most delicate manipulation should be practised.*

Methæmoglobin in the Urine; Hæmoglobinuria.—This is a condition characterized by the appearance of the coloring-matter of the blood without red-corpuscles in the urine. While it is generally known as hæmoglobinuria, the coloration of the urine is due not to hæmoglobin, but to methæmoglobin.

The urine presents a dark color, which has been compared to that of porter, strong tea, or port wine. It is usually turbid, and deposits a chocolate-colored sediment. It is always strongly albuminous. Microscopically, the sediment is found to consist of methæmoglobin, hyaline casts, and crystals of hæmatoidin, uric acid, and oxalate of lime. Blood-cells are few in number or entirely absent. The fact that the abnormal appearance of the urine is due to blood-coloring matters must be determined by the chemical blood-tests of Heller and Almen, already described.

Hæmoglobinuria is due to a disintegration of the red-blood cells, by reason of which their coloring-matter is discharged, together with an inability on the part of the liver to take care of it. It may be brought about by a great variety of conditions, as follows :

1. The transfusion of the blood of animals into that of ma
2. The intravenous injection of pure water or glycerin.
3. Extensive burns.
4. Numerous infectious diseases, notably
   - a. Scarlatina.
   - b. Variola.
   - c. Yellow fever.
   - d. Malarial fevers.
5. Blood diseases.
   - a. Chlorosis.
   - b. Icterus gravis.
   - c. Leucocythæmia, with cholæmia.
6. Certain poisons, including
   Arseniuretted hydrogen, sulphuretted hydrogen, chlor potash, quinine (in malarious subjects), pyrogallic naphthol, nitro-benzol, and poisonous mushrooms.

The most interesting cases of hæmoglobinuria are those class paroxysmal, and which may be said, in the present light of pathology, an idiopathic disorder. The causes of this disease are generally fou exposure to cold, Raynaud's disease, over-exertion, and syphilis. are affected more frequently than females. Individual attacks are us in with a rigor, followed by fever and the characteristic changes i urine. The individual paroxysms last from three to twelve hour may be repeated at intervals of a few hours to several months. The and spleen are found swollen and tender. Urticaria sometimes follow attacks. With the subsidence of the diagnostic symptoms the patien exhausted.

Chyluria.—In this condition the urine presents a milky appea After standing for a time it may undergo coagulation, by reason of numerous fibrinous masses appear, or the entire fluid may be con into a gelatinous mass.

Microscopically, chylous urine is found to contain numerous fat-ules, which show themselves as highly-refracting globules with dar lines, soluble in ether, chloroform, or bisulphide of carbon. Usuall condition is associated with leucocytes and albuminuria. Red-blood are sometimes present, and, when in considerable quantity, impart t urine a pinkish color. Very often, too, tube-casts may be found.

Chyluria occurs almost exclusively in the tropics or in person have resided a long time in those regions. It is found in associati most cases, with the filaria sanguinis hominis in the blood. Exceptio however, its parasitic origin cannot be traced, and then, according to C it may be due to changes in the renal parenchyma. The avenue by the chyle enters the urine has been a subject for considerable discu

Grim contends that chyluria is dependent upon an unnatural communication between the lymphatics and the urinary passages.

Cases have been reported in which the chylous urine underwent partial coagulation while still in the bladder.

Fat in the Urine; Lipuria.—A small quantity of fat enters into the composition of normal urine, but so small, indeed, as to be of no clinical importance. Physiologically, fat may appear in the urine in children after the ingestion of cod-liver, castor, or olive oil. Small quantities of fat may be observed pathologically in cases of chronic parenchymatous nephritis, phosphorus poisoning, and in diabetes mellitus.

Urine containing an abnormal quantity of fat presents a turbid appearance, which disappears on shaking up with ether. Under the microscope, the fat may appear as globules or as needles,—the latter especially in chronic nephritis and septicæmia.

Uroerythrin.—Uroerythrin is one of the normal coloring-matters of the urine. It is this substance which gives to uric acid and its compounds their characteristic color. It occurs in excessive quantity in cases of relative or absolute insufficiency of the liver cells; also in acute febrile diseases, and in chronic diseases of the heart and lungs.

The following test has been suggested: Precipitate the urine with the neutral acetate of lead, or with barium chloride solution. If uroerythrin is absent, the precipitate will be milky white; if in appreciable quantity, a pale-rose color; if in excess, the precipitate will exhibit a pronounced rose color.

The presence of a pinkish or reddish color to a deposit of urates demonstrates the presence of uroerythrin.

In most cases in which uroerythrin is in excess, the same fact will obtain as to urobilin.

Urobilinuria, or Excess of Urobilin in the Urine.—Urobilin is present in small quantity in normal urine, and may exist as a pigment in large quantities in pathological urine. It is a reduction product of bilirubin; and, according to von Jaksch, the latter substance may be found in the blood in almost every case in which urobilin is present in the urine. It is regarded by von Jaksch as "valuable evidence of hepatic disease when supported by other symptoms, and in the absence of causes which are known to produce it." Its occurrence also seems to bear some relation to hæmorrhagic extravasations, as urobilinuria has been observed as a prominent feature of intracranial hæmorrhage, hæmorrhagic infarctions, retro-uterine hæmorrhages, the extensive cutaneous hæmorrhages due to scurvy, hæmophilia, etc. It has also been found in hepatic cirrhosis, chronic malaria, pernicious anæmia, carcinoma, scarlet fever, pneumonia, erysipelas, typhoid fever, acute sepsis, appendicitis, acute articular rheumatism, and lead-colic.

Test.—To a test-tube about half-full of the suspected urine add five or six drops of a 10 per cent. solution of zinc chloride; next add sufficient liquor ammoniæ to dissolve the precipitate. The presence of urobilin is demonstrated by a greenish fluorescence which is best seen when the tube is held against a black background.

Urorosein.—This occurs in the urine as a chromogen, but only in small amounts under normal conditions. It may be greatly increased in pulmonary tuberculosis, penicious anæmia, and chlorosis, especially; also in diabetes, osteomalacia, typhoid fever, abdominal carcinoma, nephritis, and diseases of the stomach.

Test.—Add 15 drops of strong hydrochloric acid to 10 c.c. of urine in a test-tube. If urorosein be present in excess, there will appear a rose-red color in about ten minutes. This reaction may be accelerated by heating the mixture.

Hæmatoporphyrin in the Urine, or Hæmatoporphyrinuria.—Traces of this substance may be found in normal urine, but not sufficiently large to be practical in clinical analyses. When present in excess, the urine presents an opaque, almost black color to reflected light; in mild cases, the color may range from light-orange to Burgundy red. Uncomplicated hæmatoporphyrinuria is never associated with albuminuria. Great uncertainty surrounds the significance of this symptom; hence, for the present, it cannot be regarded as possessed of much clinical importance. It has been observed in gout, acute inflammatory rheumatism, chorea, tuberculosis, Addison's disease, cirrhosis of the liver, and after repeated doses of sulphonal and trional. Nakarai claims it to be a constant phenomenon of lead-poisoning. As a transitory symptom it is said to be of frequent occurrence in typhoid fever, and Stokvis has shown that it may result from the absorption of blood from the alimentary canal and its subsequent elimination.

All tests are unsatisfactory excepting that with the spectroscope, an this can hardly be available for the general practitioner. Von Jaksch suggests the following chemical

Test: Precipitate 30 c.c. of the suspected urine with alkaline solutio of barium chloride. Then wash the precipitate with water. Next suspen this precipitate in absolute alcohol. The addition of acids or alkalies wi take up the red color from the precipitate.

Bile in the Urine.—The elements of the bile which may be foun in the urine are the bile-acids and the bile-pigments. The latter impart the fluid a yellow, yellowish-green, green, or dark-brown color, accordir to the quantity present. Organized sediments found in the urine, as cast epithelium, etc., are stained yellow.

Choluria occurs in all conditions producing jaundice. It may also associated with diseases of the liver not attended by jaundice. It is prese in cases of obstruction of the biliary ducts, whether due to a swelling of the mucous membranes of the same, biliary calculus, or tumors of the liver and adjacent organs. It has been observed in certain conditions of the blood characterized by hæmorrhage into the tissues, and in poisoning by phosphorus and the aniline dyes.

Usually the bile-acids and -pigments are associated; hence the test for the latter, being more readily applied, answers most clinical purposes. According to Oliver, however, the biliary acids precede the pigments in their advent, and remain for some time after the latter have disappeared.

Ogden teaches that the bile-acids are not generally present in the hæmatogenous form of jaundice. He admits, however, that this rule is subject to a sufficient number of exceptions to make the presence of the bile-acids an unreliable sign of the hepatogenous origin of the jaundice.

The biliary pigments are four in number, as follows: Bilirubin, biliverdin, biliprasin, and bilifuscin. The first of these is the only one found in freshly-voided urine, the others appearing only on standing. Hence, in testing for bile in the urine, we practically limit our investigations to the discovery of bilirubin.

Urines containing bile are practically always albuminous. Owing to the irritating effect of the bile on the urinary tubules, casts and renal epithelium cells are commonly present. These anatomical elements are usually bile-stained.

Gmelin's Nitroso-Nitric-Acid Test.—To a portion of the suspected urine in a test-tube, some fuming nitric acid is carefully added by the contact method. If bile is present, there will appear at the line of union of the two fluids a play of colors, as follows: green, blue, violet, red, and yellow. Of these, green is the one color necessary. Fuming or nitroso-nitric acid may readily be made from the ordinary acid by adding some sugar to the same and boiling.

A very convenient modification of Gmelin's test is that of filtering some of the suspected urine through filter paper. A drop of fuming nitric acid is then applied, and there will appear a play of colors surrounding the drop of acid.

Ultzmann's Test.—Ten c.c. of urine are treated with 3 or 4 c.c. of a strong potassium hydrate solution, and then acidulated with hydrochloric acid. If bile-pigment is present, a beautiful green color will appear.

Iodine Test.—An alcoholic dilution of tincture of iodine 1 to 10 is applied to the suspected urine by the contact method. A green ring will appear at the point of contact, if bile is present.

Tests for Bile-Acids.—According to Oliver, the bile-acids may be detected in normal urine in the proportion of 1 : 15,000. They are found to be diminished in so-called bilious attacks. They are increased in febrile disorders, hepatic conditions, including congestion, cirrhosis, amyloid degeneration, and acute yellow atrophy.

The most accurate test is that proposed by Oliver. It is as follows: The urine must be carefully clarified by repeated filtrations, and rendered acid in reaction if alkaline. Its specific gravity must be reduced to 1008. Twenty minims of the urine are then added to the one drachm of the following test-solution:

Pulverized peptone (Savory and Moore's),	30 grains.
Salicylic acid,	4 grains.
Acetic acid,	30 minims.
Distilled water, sufficient to make	8 fl. oz.

If the bile-acids are in excess, there will appear a milkiness proportionate in density to the amount in the urine. If but normal in quantity, the turbidity will appear only after a short time. The cloudiness thus produced disappears on shaking the mixture, but may be caused to reappear by adding more of the reagent.

Another test is the following: Dissolve a little cane sugar in the urine. Dip into it a piece of filter paper, which is then set aside to dry. Then a drop of strong sulphuric acid is applied to the paper, and in a few seconds a violet color, best seen by transmitted light, appears at the point of contact. Care should be taken not to use too much sugar, otherwise the sulphuric acid will stain a brown color.

Ehrlich's Diazo-Reaction.—The reagents required for this reaction are as follows:

(*a*) Sulphanilic acid,	2 grammes.
Hydrochloric acid,	50 c.c.
Distilled water,	1000 c.c.
(*b*) 0.5 per cent. solution of sodium nitrite.	

To obtain the reaction, take 50 parts of (*a*) and mix with 1 part of (*b*) in a test-tube. Then add an equal quantity of the urine. Next, 1 c.c. of liquor ammonia is added by the contact method. If the diazo-reaction is present, there will appear a pink or red ring at the line of junction of the two fluids.

The diazo-reaction appears in typhoid urines at about the end of the first week, at which time it is rarely absent. It was at one time claimed that it was a reliable sign of typhoid fever. It is now known, however, that it may appear in acute tuberculosis, tubercular meningitis, measles, rheumatism, nephritis, pneumonia, arthritis, cirrhosis of the liver, septicæmia, and other diseases.

Ethereal Sulphates.—The ethereal sulphates which may be founc in the urine are *indoxyl potassium sulphate* and *phenol potassium sulphate* Of these, the former, generally known as indican, is especially important.

It has been definitely decided that indican appears in small quantities in normal urine, and that an increase (**indicanuria**) is fairly conclusive evidence of putrefactive processes somewhere within the bodily economy. In healthy individuals, the largest quantity is observed in those subsisting mainly upon an animal diet, particularly of red meats; and the smallest quantity in those on a milk or kefyr diet. *Pathologically*, it is found to be increased in cases of intestinal putrefaction. Inasmuch as diminished gastric acidity leads to this condition, an increase of indican goes hand in hand with a diminution of the hydrochloric acid secretion in the gastric juice. Indicanuria is especially well-marked in gastric carcinoma, the only condition presenting the phenomenon to a more marked degree being cases of ileus. Interference with the peristaltic action of the small intestines surely leads to indicanuria; and especially does this remark apply to ileus. Indicanuria from this cause is entirely independent of the amount of the gastric secretion. This is an important point to remember, for indicanuria does not occur in cases of simple, uncomplicated constipation, nor is it present in obstruction of the large intestine. Lastly, indicanuria occurs in putrefactive processes outside of the intestinal canal, *e.g.*, in empyema, putrid bronchitis, pulmonary gangrene, advanced phthisis, etc.

Tests for indican, to be of practical use to the physician, can be approximative only. The most convenient is the following: To equal parts of urine and hydrochloric acid in a test-tube add a few drops of a freshly-prepared solution of hypochlorite of soda. Next add 1 to 2 c.c. of chloroform. The mixture is then set aside for a time. Indigo is set free in the reaction and taken up by the chloroform, which becomes of a blue color, the intensity of which indicates the extent of the indicanuria. The presence of bile-pigment interferes with this test; so, when this is present, it should be removed by precipitation by a solution of subacetate of lead, which must not be in excess.

Bury recommends the following: To 2 c.c. of urine and 2 c.c. of chloroform, 1 c.c. of peroxide of hydrogen is added. Then 2 c.c. of hydrochloric acid are added. The tube is warmed, gently turning it on its axis. The chloroform is turned blue by the indigo which is formed.

Ogden recommends the following: "Take 15 c.c. of strong hydrochloric acid in a wineglass, add one or two drops of strong nitric acid, stir, then add thirty drops of the urine to be tested, and stir immediately. An amethyst color soon makes its appearance, reaching its greatest intensity in from five to twenty minutes. The amount of color obtained at the point of greatest intensity furnished some data as to the amount of indoxyl present. If *normal*, a distinct but not intense amethyst color appears; if *increased*, the color is decided, and very deep; and if *diminished*, there will be but very little color, and rarely an entire absence of color."

Phenol Potassium Sulphate.—This possesses the same significance as indican. The test for it is as follows: Acidulate the specimen of urine with sulphuric acid. Then distill, and phenol will appear in the distillate, where its presence will be made known by the addition of bromine-water, which will produce the yellow crystals of tribromphenol. Or the distillate may be warmed with Millon's reagent, and, if phenol be present, a cherry-red color will appear; or ferric-chloride solution may be added to the distillate, and a deep violet color results.

Uric Acid.—To the writer the current views concerning the pathology of uric acid are in sad need of revision. That an excess of uric acid within the body is a serious evil there can be no doubt; that the quantitative presence of uric acid in the urine constitutes important evidence of disease has been far from proven. It is even a question whether uric acid *acts* as a direct poison or by reason of its great insolubility, or, again, by reason of forming certain compounds which are deposited in the tissues, principally in the joints.

Uric acid is a normal constituent of the urine, from one-half to one gramme being excreted in the course of the twenty-four hours. It is very soluble in water, 1 part of uric acid requiring 15,000 parts of water for its solution. It is more soluble in urine rich in salts and coloring matter, hence in concentrated urines. It is liable to precipitation in dilute urines; hence a uric-acid deposit under such circumstances cannot be regarded as evidence of excess of that substance. Knowing, as we do, that neurotic subjects excrete large quantities of pale, watery urine, we can well question the relation of cause and effect of the uric acid to the nervous symptoms.

There are those who hold that it is not the absolute quantity of uric acid excreted daily that is of importance, but its relation to the excretion of urea. Haig contends that the proportion of uric acid to urea should not exceed 1 to 33.

Physiologically, it is increased by a nitrogenous diet, by rest and sedentary habits, by defective action of the liver, and, what amounts to the same thing, a diet which overtaxes the hepatic capacity. Pathologically, it is increased in fevers, inflammations, diseases which diminish respiration, leucocytosis, pernicious anæmia, ague, certain cases of diabetes mellitus, and splenic leukæmia. Administration of the salicylates increases both the production and elimination of uric acid, and the same effect is said to result from substances rich in nuclein.

Uric-acid secretion is diminished in the acute stage of gout, advanced cases of nephritis, arthritis, and chlorosis, and many cases of chronic disease.

Strong claims have been made for increased uric-acid formation and excretion on a diet of meat; but this point cannot be accepted as positively proven, for the animal diet produces increased acidity of the urine, and this, in turn, brings about more ready precipitation of the contained uric acid.

Careful quantitative analyses are, therefore, necessary to reach positive conclusions.

The presence of uric-acid crystals in the urine does not necessarily imply excess. They are always found in urine that has been standing for twelve or more hours. If, however, this substance appears in urine within six hours, there is something wrong. This precipitation is often a more important matter than excess in quantity, for it is the insolubility of uric acid that leads to much of the disturbance which it occasions.

The primary origin of uric acid is not altogether clear. We know that most of it originates in the liver, by reason of defective action of that organ; and also that some is formed in the spleen. Beyond these facts we can say nothing.

The methods proposed for the discovery of uric acid are both chemical and microscopical.

The Murexid Test.—This is a very delicate test, but it affords no other information than the presence of uric acid. To a small quantity of the urinary sediment a few drops of nitric acid are added. The mixture is then heated in a flame until almost dry. Then a few drops of ammonia are added. In the presence of uric acid a purple color will appear.

Heintz's test for the quantitative determination of uric acid is a simple one, but is uncertain, because uric acid does not always crystallize out of the solutions. Two hundred c.c. of urine are filtered, and 10 c.c. of hydrochloric acid added. The mixture is then set aside for twenty-four hours, during which time the uric acid will separate in crystals. These are then carefully collected and thrown on a filter of known weight. After washing the crystals thoroughly in cold water, and drying, the filter is again weighed. The difference in the two results gives the quantity of uric acid in 200 c.c. of urine.

Hopkins's method is now regarded as the most satisfactory for clinical work. To 100 c.c. of urine, ammonium chloride is added to saturation. About 30 grammes will be sufficient for this purpose. The solution is then permitted to stand in a cool place for 24 hours. Then filter, and wash the filter with a saturated solution of ammonium chloride. Next, the precipitate is washed off the filter into a porcelain capsule with a stream of hot water, and treated with an excess of hydrochloric acid (5 c.c.), heated to boiling, and set aside in a cool place for 2 hours longer. "Then, if the fluid has become cold, filter and wash with cold distilled water. Before washing, however, measure accurately the filtrate, as 1 mgm. must be added to the final result for each 15 c.c. of the filtrate. This need not, however, exceed 20 to 30 c.c.

"Wash the uric acid from the filter with a little hot water, add a little sodium carbonate, and heat until the crystals are dissolved. Add enough water to make 100 c.c., transfer to a flask, mix it with 20 c.c. of pure concentrated sulphuric acid, and immediately titrate with a one-twentieth normal solution of potassium permanganate solution, constantly agitating the fluid. As soon as a pink color begins to appear, titrate slowly, until the color becomes permanent. The standard N-20 solution contains 1.577 gm. of potassium permanganate in a litre of distilled water, and 1 c.c. represents 0.00375 gm. of uric acid."

Von Jaksch recommends that the uric acid be estimated by weight, instead of by titration, as he regards it a more accurate procedure. For this purpose, the solution, after evaporation with hydrochloric acid, is placed in a Ludwig filter of known weight.

Amorphous Urates.—The amorphous urates are compounds of sodium, potassium, ammonium, and calcium and uric acid as quadriurates, the quadriurate of soda being decidedly the more abundant. They are stained by the uroerythrin a fawn to pink color, in most instances, although in children they may be perfectly white. A urine rich in urates often presents a purplish film on the surface or sides. Uratic urine is usually clear when passed, the deposit appearing on cooling. The quadriurate is readily decomposed by the addition of water, converting it into the biurate and uric acid, the latter appearing as characteristic crystals.

The amorphous urates are recognized clinically by a heavy deposit, as described above, disappearing on heating the urine. They are found in cases of illness associated with fever, in functional disorders of the stomach, diseases of the viscera associated with emaciation, cirrhosis, and other disorders of the liver.

The microscopic appearance of the urates, as well as a consideration of the crystallized urates, will be considered when treating of the microscopic examination of the urine.

Xanthin.—This substance is closely related to uric acid. It is found in very small quantity in normal urine, Neubauer discovering only one gramme in 300 litres of urine. Others, however, have claimed its presence in about one-tenth the quantity of uric acid. It is said to occur occasionally in crystalline form in the urine. Simon states, however, that the majority of such cases are in reality examples of colorless crystals of uric acid. It is said, also, that xanthin may be a constituent of a very rare form of calculus.

Xanthin is increased in leukæmia, in some fevers, and in certain nervous diseases, but especially in cervical hypertrophic meningitis and tabes dorsalis.

To test for the presence of xanthin, mix a few drops each of the urine and nitric acid in a porcelain capsule, and evaporate to dryness. In the presence of xanthin there remains a yellowish deposit, which is changed to red by a potassium hydrate solution, and made reddish by heating.

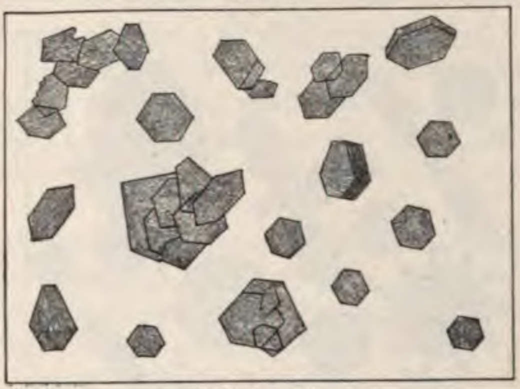
Heteroxanthin is found in normal urine only in infinitesimal quantity. It occurs in excess in leukæmia and phosphorus poisoning, and after the ingestion of theobromine and caffeine.

Test.—The addition of hydrochloric acid and a trace of potassium chlorate to the suspected urine gives a red precipitate, which changes to a violet on the addition of potassium hydrate solution.

Hypoxanthin occurs in the urine in excess in cases of leukæmia, in diseases of the liver and kidneys, fevers, and diseases of the nervous system.

Cystin.—Cystin is very rarely observed as a urinary constituent. Its chief clinical interest lies in its association with calculi composed of it. Still it may occur independently, though in such instances no symptoms evidencing a pathological process may appear for years. Cystinuria has been observed in chlorotic women, strumous children, numerous disorders of the liver, advanced renal disease, acute articular rheumatism, and as a family condition.

FIG. 115.



Cystin crystals.

Urine containing cystin presents a pale and oily appearance. It has a peculiar odor, which has been likened to sweet-brier. It is very apt to undergo decomposition, evolving sulphuretted hydrogen. When freshly voided it is turbid, and deposits a light-colored sediment.

Under the microscope it appears as six-sided opalescent tablets, which may appear singly or arranged in clusters. They may be dissolved by ammonia, hydrochloric acid and oxalic acid.

Leucin and Tyrosin.—The chief clinical value of leucin and tyrosin in the urine arises from their relation to acute yellow atrophy of the liver. While it has been claimed that they may appear in the urines of small-pox and typhoid fever, Simon takes the ground that they are almost pathognomonic of acute yellow atrophy of the liver,—the only exception he makes to this sweeping statement being cases of phosphorus poisoning, with hepatic atrophy. These substances usually remain dissolved in the

centrated sulphuric acid, and immediately titrate
normal solution of potassium permanganate sol
the fluid. As soon as a pink color begins t
the color becomes permanent. The stan
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Von Jaksch recommends
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From a practical standpoint, these may
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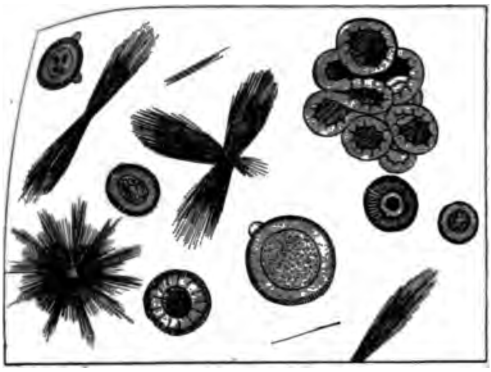
total quantity eliminated in twenty-four hours is
They are derived from muscular tissue, either
consisting of meat, or because of general wasting of

FIG. 116.



Leucin (spheroids) and Tyrosin (tufts, sheaves and stars).

tissue. They are therefore increased by a diet of nitrogenous food, in the *pyrexia* of acute diseases, diabetes mellitus, and in the advanced stages of *diseases* of the kidneys.

To test for creatinin, acidulate the urine with nitric acid, and then add phosphomolybdic acid. In the presence of creatinin, there will appear a yellow crystalline deposit which is soluble on the addition of hot nitric acid.

Zinc chloride reacts with creatinin to form rosettes of needle-like crystals.

Hippuric Acid.—The presence of hippuric acid in the urine possesses no practical clinical interest, especially as the tests for its detection are tedious and time-consuming.

Nothing definite is known concerning its absence from the urine though such a condition has been observed in acute and chronic parenchymatous nephritis after the administration of benzoic acid, the latter appear n the urine unchanged. It has been found in excess in acute febrile rs, hepatic diseases, diabetes and chorea.

siologically, the quantity of hippuric acid is increased by a vege- and by the administration of benzoic acid and salicylic acid. It l by an exclusive meat diet.

—Urea constitutes about one-half of the total solids of normal it is, moreover, the most important of the excrementitious sub- ces discharged by the kidneys. Owing, however, to the many condi- ions which may influence its quantity, its diminution or increase must be interpreted with care, and only after studying all the associated clinical phenomena.

The average normal daily excretion of urea in the male adult is about 500 grains. It is generally estimated that 3½ grains should be excreted for each pound of bodily weight. Women excrete both a smaller gross quantity per diem, and in smaller ratio to bodily weight, than men. The absolute daily quantity eliminated by children is less than in adults; at the same time, their excretion per pound of bodily weight is much higher, even to 6 grains per pound. Beginning with middle life, urea excretion diminishes; it is estimated to be 5 per cent. less from forty to fifty years of age; and after fifty to be 10 per cent. less than the standard usually accepted as normal.

The urea excretion varies considerably from day to day in the healthy individual, under the influence of physiological conditions. Thus, a diet consisting largely or entirely of nitrogenous food effects an enormous increase, even to the extent of 100 grammes or over 1500 grains in a single day. Exercise, whether bodily or mental, has a similar influence, though to nothing like such a marked degree. The free drinking of water by its solvent powers increases the absolute quantity eliminated, while lessening the percentage per ounce in a given specimen of urine. Hot baths are capable of causing a temporary increase in urea elimination.

A pathological increase of urea is especially common in febrile disorders, being due to the great tissue-destruction by the pyrexia. It is common in the course of treatment by thyroid extract. In diabetes, increased urea elimination is the rule. It is due in part to the tissue-destruction, and to the nitrogenous diet upon which these patients so largely subsist.

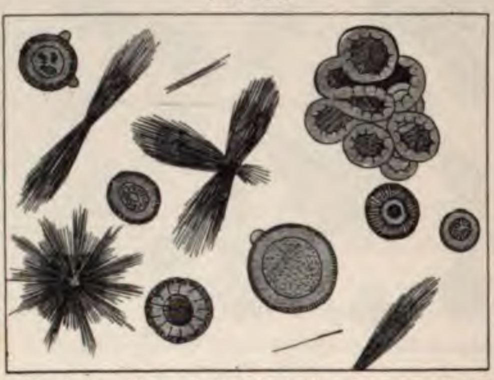
Urea elimination is diminished normally by an indolent life, as such an existence tends to retard tissue-changes; by a vegetable diet, as affording less nitrogenous waste; and after prolonged fasting. It is observed in those who are addicted to excessive use of alcoholic beverages. Inasmuch as the liver is the principal organ for the formation of urea, diseases of that viscus are necessarily associated with lessened urea excretion. Especially is this manifested in acute yellow atrophy of the liver, in which urine, but they may be deposited on slight evaporation. Tyrosin may be deposited spontaneously, in which case it appears as clusters of fine, delicate needles arranged as tufts, or in sheaf-like bundles.

Leucin appears as yellowish, highly-refracting globules suggestive of oil-drops, but differing from them in that they present concentric and radiating striæ, and are not dissolved by the addition of ether.

Bouchard has made the claim that leucin and tyrosin are found in many cases of uræmia, and that their presence is of more importance as a toxic manifestation than the absence of urea.

Creatin and Creatinin.—From a practical standpoint, these may be regarded as identical substances. Creatin appears in alkaline and creatinin in acid urines. The total quantity eliminated in twenty-four hours is usually about 1 gramme. They are derived from muscular tissue, either by reason of a diet consisting of meat, or because of general wasting of tissue. They are therefore increased by a diet of nitrogenous food, in the pyrexia of acute diseases, diabetes mellitus, and in the advanced stages of diseases of the kidneys.

FIG. 116.



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Physiologically, the quantity of hippuric acid is increased by a vegetable diet, and by the administration of benzoic acid and salicylic acid. It is diminished by an exclusive meat diet.

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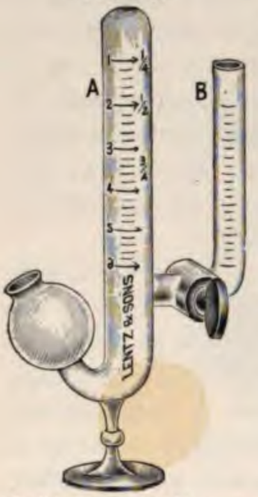
Urea elimination is diminished normally by an indolent life, as such an existence tends to retard tissue-changes; by a vegetable diet, as affording less nitrogenous waste; and after prolonged fasting. It is observed in those who are addicted to excessive use of alcoholic beverages. Inasmuch as the liver is the principal organ for the formation of urea, diseases of that viscus are necessarily associated with lessened urea excretion. Especially is this manifested in acute yellow atrophy of the liver, in which urea may be practically absent from the urine. The same remark applies to disease of the spleen, though to a much less extent. But most important of all the clinical relations of urea percentage is the daily quantity excreted in cases of renal disease, for in all forms of acute and chronic renal disorder we find the urea excretion diminished sooner or later. In acute nephritis the daily quantity is greatly diminished, though the percentage in the small amount of urine passed may be above the normal. In the chronic renal affections, the urea excretion is not affected at first. In chronic interstitial nephritis the percentage is usually low; but, owing to the polyuria, the actual quantity eliminated is not so markedly affected. Of especial interest, because such cases are very common, are those cases of nerve-prostration attended by defective metabolism, in which the urine runs a specific gravity of less than 1010, diminished urea percentage, and daily quantity of urine of, say, 30 ounces. There seems to be no evidence of organic renal disease. This condition persists for months, and yet, on the removal of such patient from home to a fine, stimulating air, a general improvement is noted.

FIG. 117.



Modification of the Doremus ureometer. The urine is placed in tube B.

All diseases and conditions characterized by lowered bodily nutrition are associated with diminished urea excretion, notably anæmia, Addison's disease, osteomalacia, and chronic rheumatism.

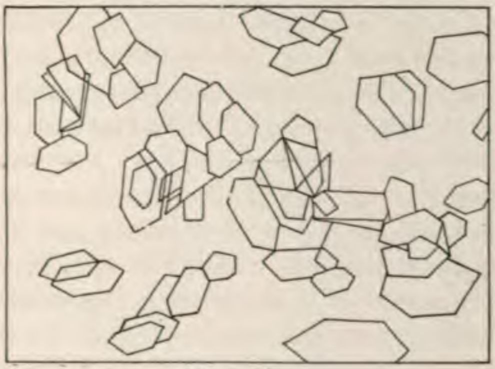
Tests for Urea.—Inasmuch as urea is always present in urine, the only useful tests for it in clinical work are quantitative. A number of these have been proposed, and are in regular use by expert chemists. Requiring time and elaborate apparatus, they are hardly practical for the busy clinician. The method to be followed in the use of the Doremus ureometer, while not strictly accurate, is sufficiently so for all practical purposes, especially as a test with it may be made in a very few minutes. The Doremus ureometer consists of an U-shaped tube, with a long and a short arm. The long arm is closed at its upper extremity, and is graduated in percentage or in fractions of a gramm according to the style of instrument selected. The short arm is widene into a bulb. With the ureometer is furnished a pipette to measure one t two cubic centimetres. (See Fig. 117.)

The test-solution of hypobromite of soda does not keep well, hence should be prepared in small quantities. A standard solution of sodiu hydrate, 100 grammes, in 250 grammes of distilled water, or six ounc to the pint, should be kept on hand. To 60 c.c. of this solution c.c. of bromine are to be added, and then diluted with an equal vo ume of water. This makes about 4 fluid ounces of hypobromite of soda solution.

To make the test, fill the long arm of the ureometer with the hypobromite solution, and then by means of the pipette insert 1 c.c. of the urine. The urine will at once undergo decomposition, nitrogen being evolved, and takes the place of the liquid in the long arm of the ureometer. After about fifteen minutes the process is completed, when the extent of the displacement indicates the percentage of urea. Platt has made an extensive series of experiments in the laboratory of the Hahnemann Medical College, showing that Labarraque's solution may be used instead of the hypobromite of soda.

The **differential density method** for the quantitative determination of urea is both simple and convenient. It is based on the facts that urine undergoes a loss of specific gravity on the decomposition of its contained urea, and that urea is decomposed by hypochlorite of soda. Every degree of specific gravity lost corresponds to 0.77 per cent. of urea, or three and a half grains to the ounce. The specific gravity of the urine is first taken. Then to one volume of urine are added seven volumes of Labarraque's solution, the specific gravity of which has also been determined. Decomposition begins at once, and is completed in the course of a couple of hours. The specific gravity of the mixture is then taken. To determine its specific gravity before decomposition, a little calculation is necessary, as follows: Multiply the specific gravity of the Labarraque's solution by 7; add the specific gravity of the urine, and divide the result by 8, and the specific gravity of the mixture is obtained. Subtract this sum from the specific gravity after decomposition, and multiply the difference in degrees by three and a half, and we have the number of degrees of urea per fluid ounce of urine.

FIG. 118.



Nitrate of urea.

To determine the mere presence of urea, it is only necessary to place a drop of urine on a glass slide, add a drop of pure nitric acid, and evaporate in the air. Crystals of nitrate of urea will be formed, and may be recognized as such under the microscope.

Chlorides.—The chlorides appear in the urine normally to the amount of 10 to 15 grammes daily. Chloride of sodium is decidedly the most abundant, but the salts of potassium, ammonium, calcium and magnesium are also present. The quantity varies greatly, according to the amount of food taken and the extent to which common salt enters into its composition. In patients who have been starved, or who have taken but little salt with their food, the chlorides are deficient or nearly absent.

Pathologically, the **chlorides are found nearly or entirely absent** from the urine in diseases characterized by exudation; hence in croupous pneumonia and pleurisy with effusion. A considerable diminution has been observed in numerous febrile disorders, due to a variety of factors, including the diminished ingestion of salt, a retention of chlorides in the blood, the diminished excretion of urine, and the elimination of chlorides by other channels than the kidneys,—as by the bowels in case diarrhœa is present. The chlorides have also been noted deficient in quantity in all varieties of renal disease characterized by albuminuria, carcinoma of the stomach, hyperchlorhydria accompanying gastric ulcer, melancholia, idiocy, dementia, chorea, and pseudo-hypertrophic paralysis.

The **chlorides are increased** in all conditions in which there has been a previous decrease by reason of exudation, and the latter is being absorbed. Hence their increase in cases of croupous pneumonia, pleurisy with effusion, and dropsy, is to be regarded as a favorable sign. They are also increased in diabetes insipidus, and in the polyuria following epileptic attacks. Indeed, the continued excess of chlorides in the urine may be regarded as a very important diagnostic sign of diabetes insipidus, especially if we can exclude the possibility of other conditions.

The extent to which chlorides are absent from the urine in febrile affections may be regarded as affording valuable prognostic indications. Cases in which they are present in less quantity than 0.05 gramme per day mus be regarded as having a very grave outlook.

Test for Chlorides.—The specimen of urine is first acidulated b the addition of a few drops of nitric acid, to prevent the precipitation c phosphates. Then a drop of a 10 per cent. solution of nitrate of silver i added. If the chlorides are present in normal amount, a thick curdy pr cipitate of chloride of silver will be thrown down. A little experience wit the test will enable the physician to judge in a general way of the extent departures from the normal standard.

The ordinary methods for the quantitative estimation of the chlorid are too technical and time-consuming for clinical work. Purdy's centrif gal method alone is practical. The graduated tube of the centrifuge filled with the urine to the 10 c.c. mark. A few drops of nitric acid are added. Then the tube is filled to the 15 c.c. mark with a solution of nitrate of silver (ʒj–℥j). The chloride of silver is then precipitated by revolving the tube, and the proportion of the sediment to the entire liquid noted. In Purdy's experience, 16 to 18 per cent. by bulk is about normal.

Sulphates.—About 80 per cent. of the sulphur appearing in the urine occurs as compounds of sulphuric acid, the balance consisting of the less oxidized forms of sulphur, as cystin, sulphocyanide of potassium, etc. Of the sulphates of the urine, two classes are recognized,—one the mineral sulphates, also called the preformed sulphates, and including the sulphuric acid compounds of potassium, sodium and magnesium, and the other class known as the combined or aromatic sulphates, including phenol-potassium sulphate, indoxyl potassium sulphate, indican, etc., to which reference has already been made.

The clinical importance of this subject is at present in a decidedly unsatisfactory state, owing mainly to the highly technical character of the chemical processes by which their quantity in the urine may be determined. This is unfortunate, because it is known that disturbance of the normal relation between the preformed and aromatic sulphates constitutes reliable evidence of putrefaction of proteid matters within the organism, mainly in the intestinal canal. The normal proportion of these salts of sulphuric acid is 10 : 1. In some cases of intestinal putrefaction, the aromatic sulphates may be increased so that the ratio becomes 10 : 5.

The **sulphates are increased physiologically** after the administration of sulphuric acid and its salts, a highly nitrogenous diet, and violent muscular exercise; in other words, the same causes as bring about increased elimination of urea.

They are **increased pathologically** in all cases in which there is increased tissue-destruction; hence we find them in excess in acute febrile processes, especially in croupous pneumonia and acute myelitis. During convalescence from these diseases, a diminution is observed. Increased sulphates have also been observed in leucocythæmia, diabetes mellitus, diabetes insipidus, carcinoma of the œsophagus, progressive muscular atrophy, and eczema.

The **sulphates are greatly diminished** in cases of poisoning by carbolic acid, and after the administration of its derivatives, as salol, lysol, etc. In such cases it will be found that the ethereal sulphates are correspondingly increased in quantity.

Tests for the Sulphates.—Acidulate 10 c.c. of urine with a few drops of hydrochloric acid, and add to the mixture about one-third of its volume of a 25 per cent. barium chloride solution. A milky-white precipitate will indicate a normal amount of sulphates; a heavy creamy deposit will indicate increase; and a faint cloud, a decrease.

Another method is as follows: Add an excess of barium chloride solution to the urine, and this will precipitate the mineral sulphates. Then filter, and thoroughly acidulate the filtrate with hydrochloric acid and boil. The sulphuric acid of the aromatic sulphates is at once set free, and combines with the excess of barium chloride as barium sulphate.

Purdy's centrifugal method is probably the most practical method of quantitative determination of the sulphates. The percentage-tube is filled to the 10 c.c. mark with urine. The acid barium chloride solution (barium chloride, 4 parts; hydrochloric acid, 1 part; and distilled water, 16 parts) is then added to the 15 c.c. mark. After thorough mixing, the barium sulphate thus formed is precipitated. Normally, it will be found to be 1 per cent. of the mixture.

Phosphates.—The total amount of phosphates excreted in the urine daily amounts to about 2 to 3 grammes. Phosphoric acid being tribasic, it enters into a variety of combinations, including acid, neutral and alkaline salts. Two classes of phosphates are recognized,—the alkaline, including the phosphates of soda and potassium, and the earthy, comprising the phosphates of calcium and magnesium. The earthy phosphates tend to precipitate in faintly acid, neutral, or alkaline urines. If the urine contains any free ammonia, the latter enters into combination with them, making a new compound, known as triple phosphate, or ammonio-magnesian phosphate.

The alkaline phosphates are very soluble, and hence do not appear in the urine as deposits.

A **determination of the total phosphoric-acid excretion** is often of great clinical importance. One must not depend upon the presence of a phosphatic sediment for the diagnosis of phosphaturia, for in the majority of such cases the precipitation is not due to phosphatic excess, but to alkalinity of the urine causing precipitation. Alteration in phosphoric-acid excretion can be determined only by actual quantitative analysis, practised according to the methods to be hereafter described.

Physiologically, the phosphates are increased in quantity after meals. Pathologically, they are diminished in the early stages of fevers and increased in the later stages. They are also said to be increased in certain neurasthenic states, hypochondriasis, and chronic articular rheumatism They are diminished in gout, in most acute diseases, most kidney lesions intermittent fever and pregnancy. Regarding the decrease of phosphori acid in renal lesions, it seems to the writer that this subject has not bee recognized as fully as it should be. Mitchell and Purdy, of Chicago, an Laidlaw, of New York, have emphasized the importance of deficient phosphoric acid elimination in Bright's disease, claiming that its presence i more regular than albuminuria. The phosphates are also increased in di fuse diseases of the bones. A form of phosphaturia has been described b

Tessier, and its existence verified by Ralfe, and denominated phosphatic diabetes. The condition is associated with emaciation, aching pains in the lower part of the back and pelvic region, excessive excretion of phosphates, dry, harsh skin, and tendency to boils, with morbid appetite.

Tests: The earthy phosphates are precipitated by heat in alkaline and neutral urines. On cooling, the precipitate disappears, unless the heating has been carried to the boiling-point, when it remains permanent. This phosphatic precipitate is dissipated by the addition of an acid.

A rough test for the presence of phosphaturia is the following: The addition of ammonia to the urine causes and produces a flocculent deposit of earthy phosphates. If, now, the fluid is filtered, and the clear filtrate acidulated with acetic acid and a solution of ferric chloride added, there will appear a yellowish-white precipitate of ferric sulphate, thus showing the presence of the alkaline phosphates.

The **Most Convenient Quantitative Test for Clinical Work is Purdy's Centrifugal Method.** The percentage-tube is filled to the 10 c.c. mark with urine, and then to the 15 c.c. mark with magnesium mixture (ammonium chloride and magnesium sulphate, of each one part; distilled water, 8 parts; and liquor ammonia, 1 part). The phosphates are precipitated. The sediment after centrifugalization should, in normal urines, constitute about 8 to 12 per cent. by bulk.

For the accurate estimation of total phosphoric-acid elimination, the following solutions are necessary:

1. A solution of 20.3 grammes of pure uranic oxide in 1000 c.c. of distilled water. One c.c. of this solution corresponds to 5 milligrammes of phosphoric acid.
2. Sodium acetate solution: 100 grammes of sodium acetate in 900 c.c. of distilled water, and to this 100 c.c. of acetic acid are added.
3. Saturated solution of ferrocyanide of potassium.

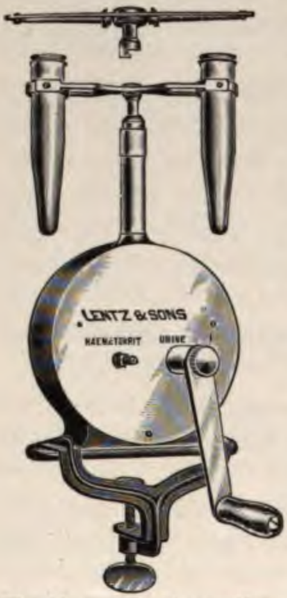
Five cubic centimetres of the sodium acetate solution are added to fifty cubic centimetres of urine, and heated in a beaker over a water-bath. Some of the uranium solution is placed in a burette and permitted to drop into this mixture, drop by drop, as long as a precipitate falls. From time to time a small portion of the mixture may be placed in a porcelain capsule and tested with the ferrocyanide solution. If a reddish-brown reaction does not take place, the mixture is returned to the beaker. The end of the reaction is determined by the complete precipitation of the phosphoric acid by the uranic salt, which is demonstrated by the reddish-brown reaction with the ferrocyanide. The quantity of uranium solution is then read off. Each cubic centimetre indicates 5 milligrammes of phosphoric acid in 50 c.c. of urine, whence may be calculated the daily excretion.

Microscopical Examination.

Proper preparation of the specimen is an important preliminary step in the microscopical examination of the urine. Sedimentation is necessary in all but the few cases in which the urine contains large quantities of solids held in suspension. For this purpose, two methods may be employed, as follows:

1. **Permitting the urine to deposit its sediment in a conical glass,** whence it may be removed by means of a pipette. This method is not the best, but it was the only means at hand until the perfection of the centrifuge for clinical work. At the present day it may be regarded as antiquated, though the only means for those who do not possess a centrifuge. It is open to several objections. Time (twelve to twenty-four hours) is required for the formation of a sediment, during which chemical changes and disintegration of the organized elements (tube-casts, etc.) may take place. These secondary changes may be obviated in a measure by the addition of some preserving fluid, preferably 20 to 30 c.c. of a solution of chloroform (7.5 c.c. of chloroform in one litre of water). Even then the method is open to the objection of delay in obtaining information. Then, too, conical glasses are difficult to clean; and it is said that the introduction of the pipette to withdraw the sediment is capable of destroying the configuration of the structures comprising the sediments. In all cases it is important that the glasses be carefully covered, to prevent the introduction of foreign materials and micro-organisms from the atmosphere. Rieder believes that cylindrical test-tubes are much more satisfactory.

FIG. 119.



The Metzger hand centrifuge, with Daland's attachment for blood examinations in position.

Sedimentation by the Centrifuge.—This method is pre-eminently the best, as it enables one to prepare the specimen while the patient is still in the consulting-room. It furthermore makes it possible to examine the urine before changes have taken place, and it makes the sedimentation much more complete than the preceding method.

Care is required in pipetting after sedimentation. The pipette use should be about one-eighth of an inch in diameter, and drawn out to point at one end, the opening of which should be about one-thirtieth of a inch. The other end should have a perfectly true flanged edge. Inasmuc as certain sediments are very brittle, and may be crushed by pipetting, a sudden rush of the fluid into the instrument is to be avoided. With this end in view, the supernatant fluid should be first poured or siphoned off so as to lessen hydrostatic pressure, and the sediment should be admitted to the pipette slowly.

When depositing the sediment on the slide, care should be taken not to deposit too much, as by so doing the lighter elements of the sediment may be driven beyond the edge of the cover-glass when the latter is placed in position. This difficulty may be avoided in great measure by proper care, and by the use of large cover-glasses, *i.e.*, an inch to an inch and a quarter in diameter.

A common error among novices is the use of too strong a light in the examination of urinary sediments. This renders the discovery of transparent objects, as hyaline casts, and certain crystals, etc., indistinguishable. Hence care should be taken to limit the light by an iris diaphragm.

Oxalate of Lime; Oxaluria.—Oxalic acid appears normally in the urine to the extent of 20 milligrammes daily. Its chief combination is with lime, forming the oxalate of lime. It is readily recognized by the microscope, appearing, as it does, in characteristic octahedra of various sizes, with highly refracting edges. More rarely it occurs in dumb-bell, hour-glass, hatchet and biscuit shapes. Modifications of the octahedral form also occur. Thus, the crystals may appear as quadratic prisms surmounted at each end by a pyramid. The anomalous forms often appear to be made up of a number of needles, thus giving them a striated appearance. They sometimes present a yellowish tinge, which, however, is never so deep as that characterizing uric-acid deposits. Some difficulty may be experienced in recognizing calcium oxalate crystals when of minute size, as at first sight they resemble leucocytes, differing only in their highly refracting power. The use of a stronger objective, however, brings out their true shape and distinguishes them.

The octahedral form of calcium oxalate can be confounded only with the crystals of ammonio-magnesium phosphate, and then only by the novice. From the phosphate they differ in their smaller size, and their regular geometric conformation. The triple phosphate, moreover, is soluble in acetic acid, while calcium oxalate is insoluble in all reagents excepting the mineral acids.

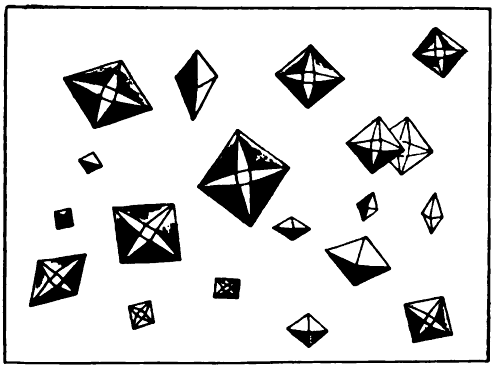
At times, the presence of calcium oxalate may be suspected by the macroscopic appearances of the sediment, the crystals, notwithstanding their small size, producing numerous sparkling points at the surface of the cloud.

The dumb-bell forms may at times resemble uric acid so closely as to make a differentiation impossible by inspection alone. Then chemical tests may be employed. Caustic soda dissolves uric acid, but has no effect on calcium oxalate.

While ordinarily transparent and colorless, oxalate-of-lime crystals may be tinged yellow in jaundice, and dark-blue by the presence of indigo in the urine.

An occasional crystal of oxalate of lime is found in normal urine. An excessive deposit may be brought about by dietetic causes, as after the ingestion of rhubarb, sorrel, garlic, grapes, oranges, the free drinking of carbonated water, and asparagus.

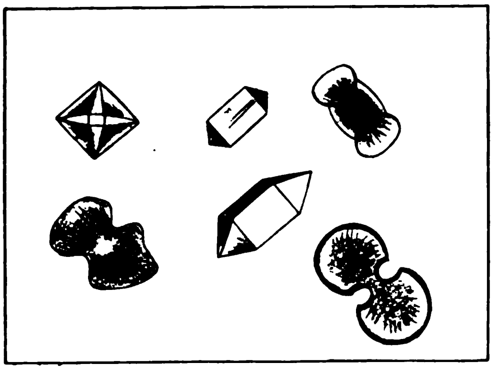
FIG. 120.



Calcium oxalate. Octahedra.

Pathologically, oxalate-of-lime deposits have been observed in neurasthenia with mild degree of albuminuria, gastric neuroses, hypochondriasis, spermatorrhœa, convalescence from fevers, and diabetes mellitus.

FIG. 121.



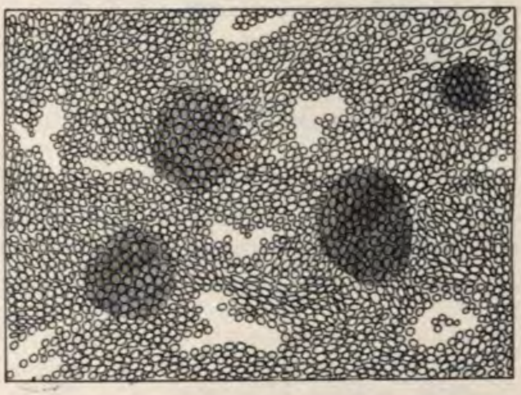
Forms of calcium oxalate.

An essential oxaluria or oxalic-acid diathesis has been described by English clinicians, the only objective symptom being the deposit of oxalate of lime in the urine. The other symptoms are emaciation, general mental and physical depression, dryness of the palms and soles, especially in the evening, irritable and excitable temper, pains in the loins, and deficient or absent sexual power. In a second variety emaciation is also present, but the digestive disturbances occupy a prominent position, the patient complaining of indigestion, flatulence, constipation, sleeplessness, rheumatic pains, and mental depression. In still a third variety there are the usual loin pains, with lancinating neuralgic pains, gastralgia, furunculosis, and nervous symptoms, but without emaciation.

Some cases of oxaluria seem to bear a relation to excessive ingestion of sugar.

The clinical status of oxaluria may be said to be in a chaotic state. In the majority of cases the condition is one of precipitation, and not excess. In still others the deposit is the result of tissue metamorphosis, and is, therefore, an expression of a constitutional condition; while, in still others, many of the symptoms are undoubtedly due to the irritating effect of the minute crystals on the mucous membrane of the urinary tract.

FIG. 122.



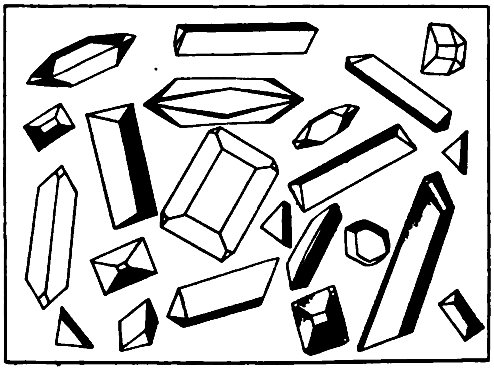
Amorphous earthy phosphates.

Phosphates.—The phosphates discoverable under the microscope are the amorphous phosphates, ammonio-magnesium phosphate (triple phosphate), and calcium phosphate (stellar phosphate).

The amorphous phosphates appear as colorless amorphous masses, consisting of granules or spheres of various sizes. They possess no clinical significance beyond indicating that the urine is in a condition to produce precipitation of the phosphates. They are readily soluble on the addition of acetic acid.

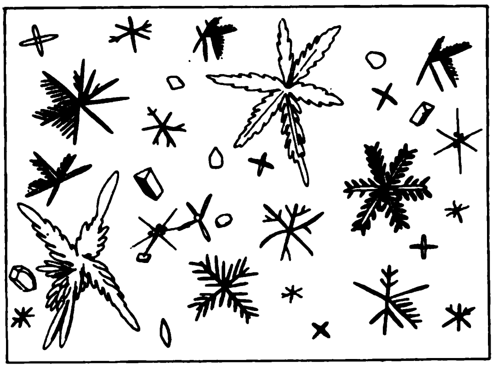
Ammonio-magnesium phosphate appears in a variety of forms. The most common is that of large crystals in the shape of three-sided prisms with bevelled ends. They have not inaptly been compared to the lid of the old-fashioned coffins. They are the largest crystals which may appear in the urine, though exceptionally they may be very small, and then may be confounded with the octahedral crystals of oxalate of lime. Less commonly the prisms are four- or six-sided, but even these have the bevelled ends. Sometimes the triple phosphate appears as feathery crystals, and may even present a strong resemblance to fern-leaves. They are transparent and colorless, unless bile-pigments are present, when they may be stained yellow. Ammonio-magnesium phosphate appears in alkaline urine which is undergoing ammoniacal fermentation. It is most commonly found in the ammoniacal urine of cystitis. It is rendered soluble by the addition of acetic acid.

FIG. 123.



Ammonio-magnesium phosphate. Coffin crystals.

FIG. 124.



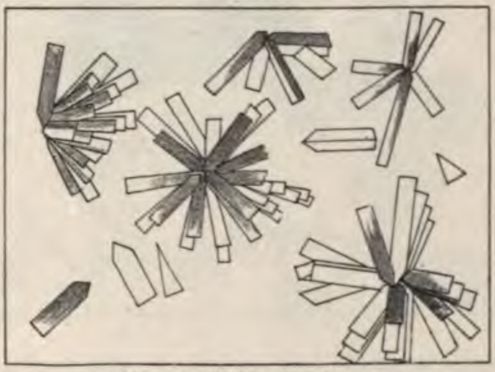
Ammonio-magnesium phosphate. Fern-leaf or feathery crystals.

Crystals of **neutral calcium phosphate** present themselves as acicular crystals with wedge-shaped or bevelled ends, occurring singly or in rosette-like groups. When very thin and needle-shaped, and arranged in tufts, they may be mistaken for tyrosin. They are colorless, and dissolve readily in acetic acid. They may occur in neutral, amphoteric or feebly acid urines. They may be found in health, but are more commonly observed in rheumatic affections of the joints, anæmia, and chlorosis.

Monocalcium phosphate is a rare form of phosphatic deposit, and is observed only in acid urines. It is usually associated with uric-acid crystals, and may be readily distinguished from the latter by its solubility in acetic acid.

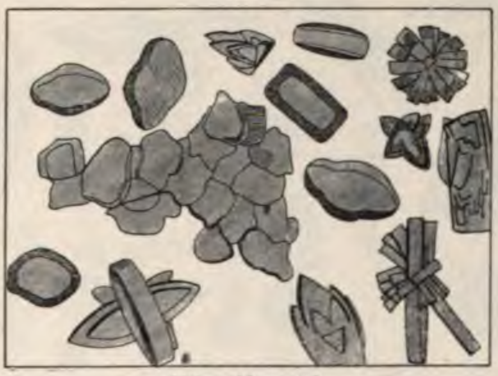
Uric Acid.—The most commonly-observed type of uric-acid crystals noticed in the urine is the whetstone-shape, occurring singly or in groups. At the same time, it must be remembered that they may appear as large rosettes, composed of tube-shaped or long, pointed crystals, rhombic plates with rounded edges, and as "dumb-bells." Usually, they are of an orange-red color; but this must not be accepted as invariably the case, for they may be colorless. The statements made by some prominent authorities that they are the only urinary crystals invariably showing color under the microscope must be taken "cum grano salis."

FIG. 125.



Neutral calcium phosphate.

FIG. 126.

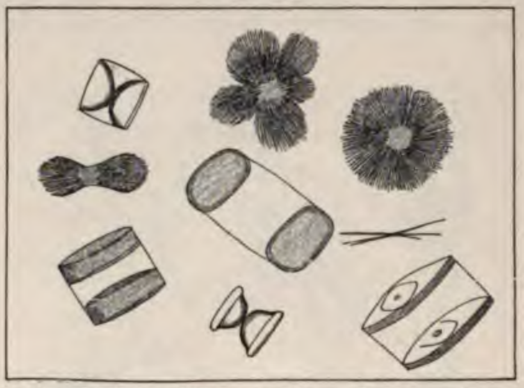


Uric-acid crystals.

Uric-acid crystals are readily dissolved by liquor potassæ, but not by acetic acid. They differ from the oxalates in being precipitated by hydrochloric acid, the oxalates being dissolved.

The clinical significance of uric acid has been referred to when referring to the chemical tests for this substance. It is only necessary to say, at this time, that such a precipitate does not necessarily indicate an excessive uric-acid excretion.

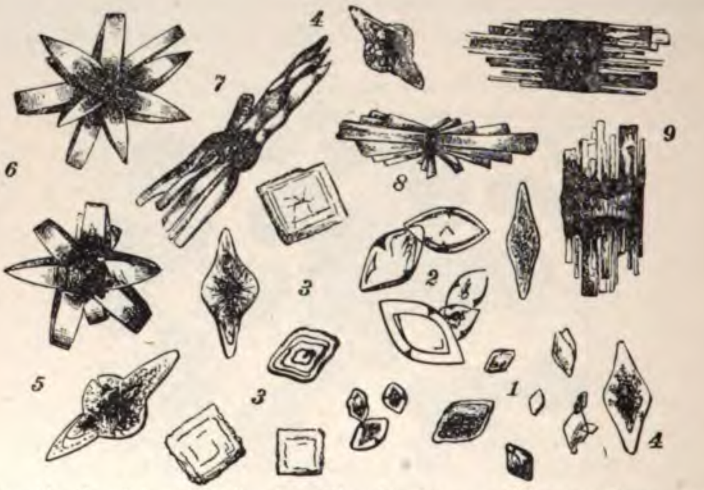
FIG. 127.



Uric-acid dumb-bell and hour-glass forms, sheaves, and rosettes.

Amorphous Urates.—These include the urates of sodium and potassium. They appear under the microscope as innumerable amorphous granules. At times they are so profuse as to obscure all other materials in the sediment. Under such circumstances other elements of the sediment may be studied by heating the urine to dissolve the urates, and then secure sedimentation by the centrifuge. Or the urine may be filtered still hot, and the contents of the filter may be washed off for microscopic examination.

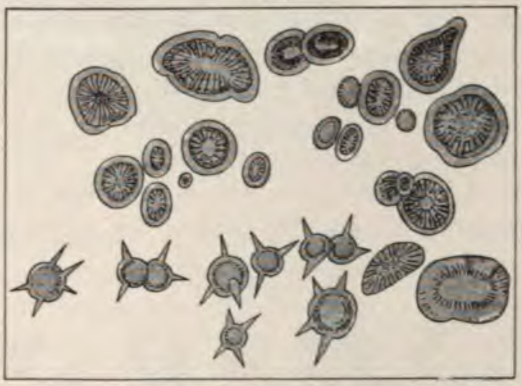
FIG. 128.



Forms of uric acid. (Landois and Stirling.) *1*, Rhombic plates; *2*, Whetstone forms; *3*, Quadrate forms; *4*, *5*, Prolonged into points; *6*, *8*, Rosettes; *7*, Pointed bundles; *9*, Barrel forms precipitated by adding hydrochloric acid to the urine.

Amorphous urates differ from the phosphates presenting a similar appearance in that they are colored by uroerythrin; they appear in acid urine, not in alkaline; and dissolve under the application of heat. The amorphous phosphates are colorless, and dissolve on the application of acetic acid. By the addition of a drop of hydrochloric acid, uric acid may be made to crystallize out as whetstone-shaped crystals. In children the urates may be colorless, in which case they may be differentiated from the phosphates by the chemical tests already referred to.

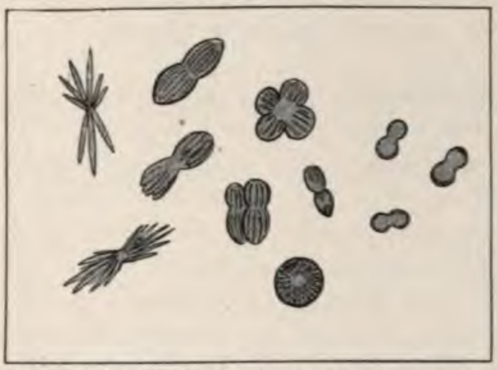
FIG. 129.



Crystals of acid sodium urate.

Sodium urate appears as irregular spheres with spiny projections. They may be deposited within the bladder, and give rise to considerable irritation of the mucous membrane of that viscus.

FIG. 130.

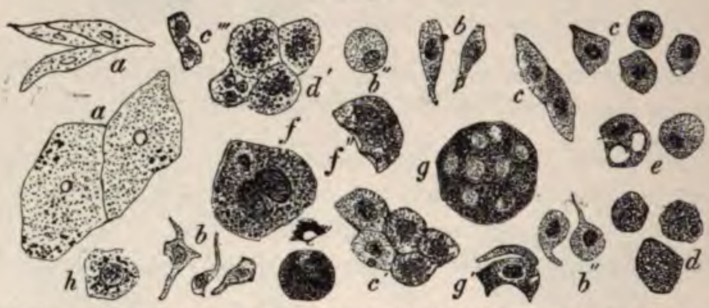


Acid ammonium urate. Spheroids, dumb-bells, double-spheroids and acicular crystals.

The **urate of ammonium** is white in color, and opaque. Its crystals, therefore, appear dark, to transmitted light, under the microscope. They have not inaptly been described as "thorn-apple" spherules. This is the only urate appearing in alkaline urine. It may occur in association with phosphatic deposits in ammoniacal urine.

Epithelium.—Epithelial cells in small numbers are found in the sediments of normal urines. When, however, they occur in profusion, they are to be regarded as pathological. To interpret their significance under such circumstances is not an easy matter, if one depends upon inspection alone, for there is hardly a type of epithelium found in one portion of the urinary tract that does not exist in the deep or superficial layers of the mucous membrane of another part. Many and elaborate attempts have been made to lay down rules for the diagnosis of the source of the epithelial cells found in urinary sediments, with the result, in the mind of the writer, of only adding confusion to the subject. The best plan of study seems to be that of Ultzmann, who has divided urinary epithelium into three classes, namely: (a) round, (b) caudate, and (c) squamous.

FIG. 131.



Urinary epithelium. (v. Jaksch.) *a*, *a′*, Squamous epithelium from the urinary sediment; *b*, *b*,′ *b″*, Epithelium from the bladder; *c*, *c′*, *c″*, *c‴*, Epithelium from the kidneys; *d*, *d′*, Fatty epithelium from the kidneys; *e–h*, Epithelium from the bladder.

(*a*) **Round Epithelium.**—These are small in size, scarcely larger than a leucocyte, differing from the latter, however, in the possession of a well-defined nucleus. At times this nucleus is scarcely visible, but is generally large, bright, round or oval. They may undergo changes by reason of the action of the urine, and hence may become swollen or polygonal. Sometimes they are small and pale, and at others large and granular. Normally colorless, they may present a yellowish or brownish hue. Sometimes they present a condition of fatty or albuminous degeneration.

These round epithelial cells are *usually* derived from the uriniferous tubules and the deeper layers of the mucous membrane of the pelvis of the kidneys. Similar cells are also found in the male urethra. When discovered in the urine, their renal origin may be inferred when a number of them are shed together, retaining the general shape of the tubule from which they were derived, and by their association with albuminuria. They ar very common in acute and chronic parenchymatous nephritis, in the latte affection often exhibiting degenerative changes. When occurring singly, renal epithelium is difficult of recognition. Their nature, then, is to be determined only by the character of the associated signs and symptoms.

(*b*) **Caudate Epithelium.**—Epithelial cells of this class present processes of various lengths and well-defined nuclei. They are derived mainly from the pelvis of the kidneys, and from the mucous membrane at the base of the bladder. The processes in the latter case are longer. It is possible to confound them with cancer-cells. The latter, when present, are larger and more numerous. This similarity is scarcely of much clinical import, because the diagnosis of renal disease will depend mostly on other symptoms and signs. In the majority of cases, the presence of caudate epithelium is suggestive of pyelitis. They are then apt to be associated with numerous round cells.

(*c*) **Flat Epithelium.**—Epithelial cells of this variety are derived from the bladder and vagina mainly ; in part, also, from the ureters and the prepuce. They are large, polygonal in shape, and have large, well-defined nuclei.

Tube-Casts.—Of all the formed elements found in the urinary sediment, the greatest interest has been attached to tube-casts. This fact seems to have originated in the difficulty attendant upon their recognition in many instances, and upon the theories provided for their formation and their clinical importance. Three theories have been suggested as to their formation, as follows :

1. That they are composed of the coagulable elements of the blood which have been transuded into the urinary tubules by reason of pathological changes in the renal structure, and have become moulded.

2. That they are composed of secretions from the changed tubular epithelium, said secretions solidifying and forming casts.

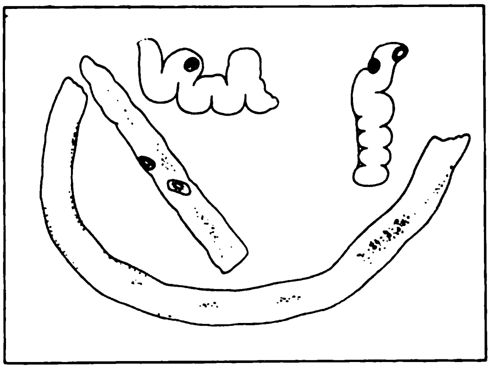
3. That they are the direct result of the disintegration of the renal cells.

Of these theories, the first is the one finding the most general acceptance.

Tube-casts as they appear in the urine may be either straight or convoluted, and exceedingly variable as to their diameters and lengths. Rieder claims that the straight casts are formed in the tubuli recti and the convoluted in the tubuli contori, but such statements do not seem to be borne out by either observation or reasoning. As a rule, their diameters are uniform throughout, excepting at one end, which is usually rounded, and therefore slightly tapering. They are very frequently broken ; hence their ends are often serrated, or "cut off short," as it were.

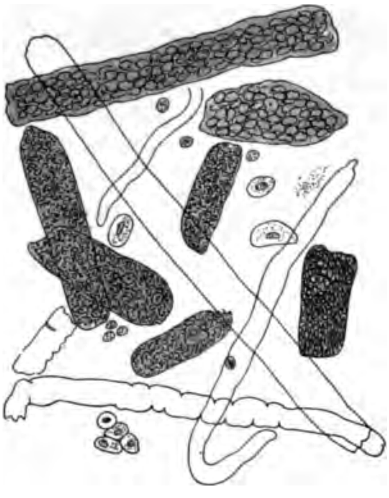
In structure they consist of a basement substance, which is known as the hyaline matrix, in which is imbedded numerous particles of granular matter. The latter is produced by degenerated epithelium. According to the size and density of the granules, these casts are divided into coarsely and finely granular. They must be distinguished from casts of similar appearance, due to a coating of amorphous urinary salts, as urates. Rieder has proposed the term "granulated" to designate these casts. The presence of epithelial cells amidst the granulations tells that they are true granular casts; and the addition of acetic acid dissolves them if they are composed of urates. Delepine, however, looks upon Rieder's differentiation as altogether valueless. In color, granular casts may vary from a yellowish-white to a brownish-red. The granules may depend upon degenerated blood-cells. The hyaline basement substance is often visible. They are most frequently observed in a fragmentary state, but are occasionally

FIG. 132.



Hyaline casts.

FIG. 133.



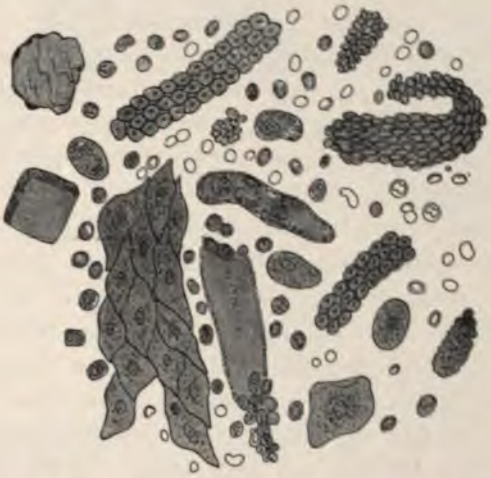
Hyaline, granular and epithelial casts; epithelial cells from the kidneys and urinary passages and leucocytes.—After Rieder.

of perfect form. They are occasionally coated with leucocytes, epithelial cells (degenerated and degenerating), fat-globules, and needles of fat-crystals.

Epithelial casts consist of the epithelium of the tubules imbedded in the hyaline matrix. Generally these cells are found in a state of granular degeneration. They are to be differentiated from pseudo-casts or epithelial tubes, in which the epithelium of the uriniferous tubules is shed *en masse*, and appears in the urine as hollow moulds.

Fatty casts are characterized by the percentage of fat-globules of various sizes adhering to the basement structure. Such casts are nearly always granular in great part. Sometimes, also, minute fat-crystals are discoverable. Some of these crystals are not soluble in ether, suggesting the inference that they are not entirely fatty in origin, but that they may consist of compounds of lime and magnesia with the fatty acids.

FIG. 134.



Blood, leucocyte and mixed casts. Epithelium from urinary passages. Uric-acid crystals.

Blood- and **pus-casts** present respectively red-blood cells and leucocytes in the substance of the structure.

Waxy casts are transparent cylindrical bodies resembling the hyaline casts very closely, but differing from them in their greater length and breadth, and in being marked by numerous indentations, suggesting segmentation. They are also tinted somewhat yellow, and are more highly refractive than the hyaline variety. Rieder speaks of them as colloid casts, a much better term than the one commonly employed, as it does not lead to incorrect clinical conceptions. They may be divided into classes, one known as the amyloid, in which they present the characteristic amyloid reaction to iodine, and the others which do not so react. They may entangle within their substance any of the anatomical elements which attach themselves to the hyaline variety, although this is not of frequent occurrence. Unlike hyaline casts, they are not dissolved by acetic acid. Sometimes they present a cloudy appearance, which has been attributed to bacterial contamination.

Pseudo-Casts.—These bear a strong resemblance to true casts, but differ from them in that they have no basement structure. They may consist of epithelium, red-blood corpuscles, or leucocytes derived from the uriniferous tubules and thrown off *en masse*. Careful inspection of those composed of epithelium show that they are hollow. The blood-casts consist of fibrin and red corpuscles.

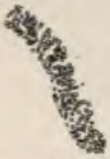
Cylindroids are long, ribbon-like bodies, presenting a uniform breadth, and, like the hyaline casts, may entangle certain cellular and crystalline elements. There is every reason to believe that they are formed in the kidneys, and are closely related to the hyaline casts.

Significance of Tube-Casts.—There was a time when the presence of tube-casts in the urine was regarded as positive proof of the existence of organic changes in the kidneys. The improved technique resulting from the introduction of the centrifuge into general clinical work has brought about a change of opinion. Glaser repeatedly found hyaline casts in the urine of persons believed to be perfectly healthy, especially when they had been subjected to some slight toxic influence, as alcoholic indulgence. Nothnagel found them in icteric urine in persons with sound kidneys. Lawrence, working in the clinical laboratory of the Hahnemann College, finds them in a very large proportion of the urines examined, irrespective of the state of the health of the patient. It would thus seem that hyaline casts, when few in number and not associated with other evidences of disease, are possessed of but little clinical importance. When, however, they are present in numbers, especially when coated with various cellular elements, they are to be regarded as pathological. Hyaline casts may occur in renal anæmia and hyperæmia, diffuse nephritis, and interstitial nephritis. In the latter affection they are, as a rule, but few in number. Authorities differ as to the prognostic value of the number of casts present, Rieder contending that there is no relation, while von Jaksch distinctly states that they are the more numerous as the extent of renal tissue involved is increased.

Granular casts are always pathological, indicating, as they do, that epithelium and blood-cells have undergone granular degeneration. They are found in acute nephritis and chronic diffuse nephritis, especially when the latter complicates a chronic interstitial nephritis. When numerous, they indicate an inflammatory condition. They are occasionally observed ir cyanotic induration of the kidneys, especially when engrafted upon a pre-existing nephritis.

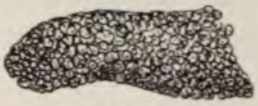
Epithelial casts necessarily indicate a desquamation of renal epithe

FIG. 135.



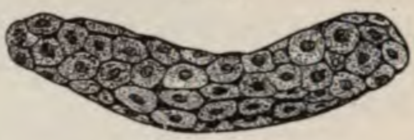
Cast of urates.

FIG. 136.



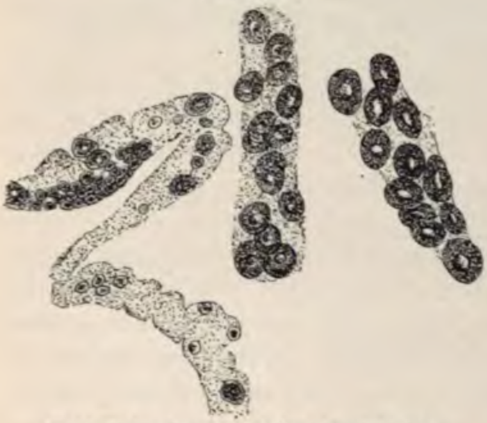
Blood-cast.

FIG. 137.



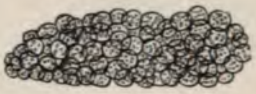
Epithelial cast.

FIG. 138.



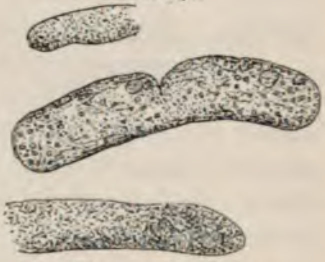
Epithelial casts, not rich in epithelial cells.

FIG. 139.



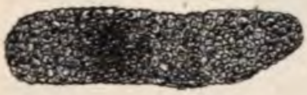
Pus-cast.

FIG. 140.



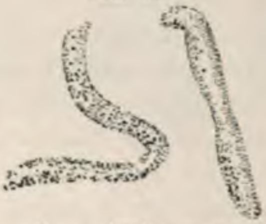
Fine granular casts.

FIG. 141.



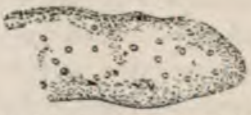
Coarse granular cast.

FIG. 142.



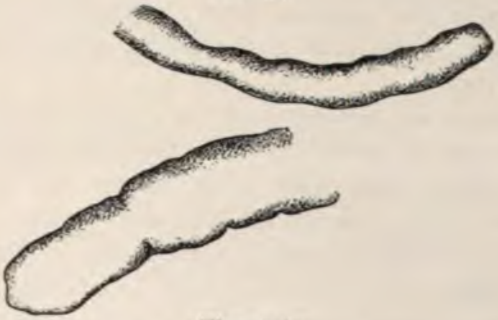
Narrow hyaline casts.

FIG. 143.



Broad hyaline cast, containing a few drops of oil.

FIG. 144.



Waxy casts.

NOTE.—Figs. 135 to 144, inclusive, from Goodno's "Practice of Medicine," are presented as representing the typical casts of the several varieties.

lium. They are especially liable to occur in acute desquamative nephritis and chronic parenchymatous nephritis. It is always important to take cognizance of the condition of the epithelium entering into the composition of the casts—that is, whether shrunken or atrophic, or whether associated with fat-globules or granular matter.

Blood-casts may arise from most renal conditions producing hæmaturia. At the same time they are more frequently observed in the course of acute parenchymatous nephritis, or during an acute exacerbation of a chronic nephritis. In the former case epithelial, and in the latter granular casts will be present in large numbers.

Pus-casts most surely indicate a suppurative condition of the kidneys.

Fatty casts occur only in cases of nephritis running a protracted course. They are observed most commonly in chronic parenchymatous nephritis. They are to be regarded as an unfavorable sign.

Waxy casts possess pretty much the same significance as the hyaline variety. They may be found in acute and chronic nephritis, and in contracted and amyloid kidneys. The fact that some of them exhibit the amyloid reaction to iodine does not indicate that they originate in amyloid disease ; for it is well known that such casts may undergo the amyloid degeneration by reason of prolonged retention in the uriniferous tubules. Their presence always means chronicity; hence they are indicative of serious lesions.

Pseudo-casts, consisting, as they do, of exfoliated epithelium or moulds of effused blood, are indicative of an acute inflammatory process.

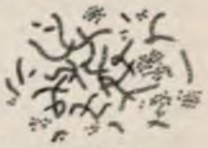
From the above remarks concerning the presence and significance of tube-casts, it is clear that they must be studied, as any other symptom, in association with the physical signs and rational symptoms, before any positive conclusion concerning their presence can be deduced. Grainger-Stewart has observed that " casts are of undoubted value in establishing the existence of Bright's disease, considered generically, but they afford comparatively little assistance in the differential diagnosis of the various forms. Any form of tube-cast may occur in any form of the disease, and at almost any stage. Indeed, all of the leading varieties may occur simultaneously in one case."

Notwithstanding the admission of these facts, Tirard closes his disquisition on tube-casts by remarking that " renal casts often afford the best and clearest indications of the nature of the changes that are affecting some part of the gland tissue, even though they do not and cannot give any clue to the extent to which the whole of the kidney is affected."

Spermatozoa.—Spermatozoa are not uncommonly observed in the urine as the result of sexual intercourse a short time before. Under such circumstances they have no pathological significance. It is not inconsistent with health to find them present in the urines of constipated subjects, the straining at stool causing the expression of semen from the seminal vesicles. They may also be found in true spermatorrhœa, following bad sexual hygiene. Such a state of affairs is of the greatest rarity.

Yeast Fungi.—Yeast fungi are sometimes found in diabetic urine of acid reaction; indeed, it has been claimed that they are never found in any other condition. It is to be admitted, of course, that when urine is sent to the physician in bottles which have previously contained some sugary substance, the ensuing fermentation may favor the presence of the yeast fungus.

FIG. 145.

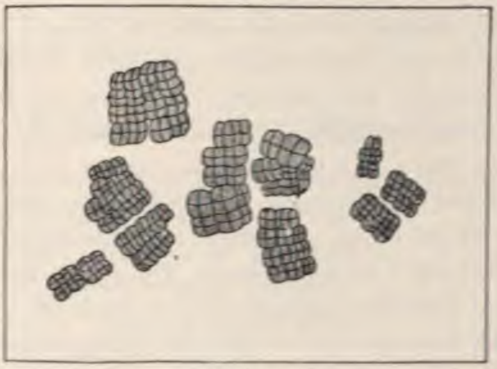


Yeast fungus. (v. Jaksch.)

Sarcinæ.—Sarcinæ are found in the urine on rare occasions only. They are similar in shape, but somewhat smaller in size than those of the stomach (*sarcinæ ventriculi*). They are without special diagnostic significance.

Micrococcus Ureæ.—This is a non-pathogenic organism appearing in urine undergoing fermentation. It appears in long, chain-like series. It occurs in almost pure culture upon the surface of the urine.

FIG. 146.



Sarcinæ.

Tubercle Bacilli.—Notwithstanding the comparative frequency of tuberculosis of the urinary passages, it is on rare occasions only that the tubercle bacilli are discovered in the urine. Hence it has been argued that negative results do not count for much in suspected cases of uro-genital tuberculosis. Deficient technique is responsible for this. Laidlaw has devised a method of procedure which unquestionably offers better results than any thus far suggested. A twenty-four hours' specimen is collected. It is then permitted to stand for about three hours for sedimentation. The bacilli are more readily found in this than in a fresh specimen. The sediment is next collected, and precipitation is aided still further by centrifugalization.

The examination may be greatly hampered by the presence of other urinary sediments. If urates are present, they must be dissolved by warming. Centrifugalization is performed in a warm tube, and the sediment is spread upon a warm slide. Any subsequent uratic deposit is gotten rid of in the washing processes attending the staining.

Phosphatic deposits are eliminated by the addition of a few drops of acetic acid.

Oxalate of lime presents a more serious obstacle. To get rid of this, spread two slides with a thick layer of sediment. When they are dry, rub them together to thin down the film. Heating over the flame will do the rest.

Pus is even more difficult to manage, and it is in purulent urines for which we most frequently search for the tubercle bacillus. For this difficulty, Laidlaw* has devised the following procedure :

" To the pus add one-fourth of its volume of liquor potassæ, and warm gently in a beaker, agitating it. The pus will coagulate and then slowly liquefy. Precipitation at the highest attainable speed will bring down a soft, bulky sediment, at the bottom of which the bacilli will be found." " It is to be remembered that too strong an alkali will destroy the bacilli, and it is sometimes impossible to find them in strongly alkalinized tubercular pus."

The sediment obtained is next spread on a glass slide. It is then ready for staining by the carbol-fuchsin method. The first drying should be spontaneous, without any warming. Fixation by heat of a Bunsen burner follows. The specimen is next covered with the carbol-fuchsin fluid, placed over a flame, and permitted to simmer gently for two minutes. " Pour off the fuchsin and immerse the slide in the nitric-acid solution, in which it loses all its red color. The time in this solution will vary with the stain, and must be judged by experience. Generally, twenty to thirty seconds will suffice. Immerse again in water to wash off the excess of acid, and cover the slide with absolute alcohol for three successive times of one-half minute each. The alcohol decolorizes the smegma bacillus, which often occurs in urine and is indistinguishable from the tubercle bacillus when stained with fuchsin. Immerse again in water to wash off the alcohol. Next cover the slide with the methylene blue for one minute. Wash off the blue, dry the slide in the air or over the flame as desired, place a drop of immersion oil on the dried film, lower the immersion lens into it, and the search for the tubercle bacillus may begin."

Gonococci.—The frequency with which gonorrhœal infection is responsible for a variety of conditions makes the discovery of the gonococcus in urethral discharges a matter of great importance. These micro-organisms stain very readily in the basic dyes, as gentian-violet, fuchsin, and methylene blue. The latter stain is generally regarded as the most satisfactory, although the cocci do not take it as readily as they do the others. It is distinguished from other organisms having similar staining reactions in its failure to stain by the Gram method. The suspected discharge is placed on a cover-slip, fixed by heat, and stained with methylene blue. This stains the gonococci and the nuclei of the pus-cells blue. The former appear as biscuit- or kidney-shaped bodies in the pus-corpuscles. The Gram method of staining requires careful attention to detail to ensure its success. It is as follows: The cover-glasses are placed in aniline-water gentian-violet solution for a couple of minutes, and then in iodine potassium iodide solution (iodine, 1.0; potassium iodide, 2.0; water, 300.0) Next, the specimen is decolorized in alcohol, rinsed in water, dried and mounted. Bacteria stained by this method assume a bluish-black color. Gonococci are decolorized.

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* *Hahnemannian Monthly*, March, 1901.

The micro-organisms of suppuration, streptococci and staphylococci may appear in the urine in connection with suppurative conditions in the urinary tract. Their identity cannot be established with certainty, excepting by culture methods.

The typhoid bacillus is present in the urine in from twenty to thirty per cent. of the cases of typhoid fever. At times they may be so numerous as to assume a diffuse cloudiness.

Disturbances of Micturition.

The disturbances of micturition include the following:

1. Simple irritability of the bladder.
2. Increased frequency of micturition.
   (a) The frequency of quantity.
   (b) The frequency of irritability.
3. Painful urination.
4. Retention of urine.
5. Difficult urination.
6. Incontinence of urine.

1. **Simple Irritability of the Bladder.**—Irritability of the bladder usually originates from disease of that organ. Nevertheless, it may arise from other than urinary causes. The influence of nervous causes in this respect is not sufficiently recognized. Just as diarrhœa may be occasioned by mental strain or excitement, so may irritable bladder result from the same cause. Unconscious imitation produces some cases. Thus, Fenwick relates the case of a student who was in the habit of walking to the hospital with a companion who was obliged to stop at short intervals to urinate. Gradually the desire to imitate the sufferer became so strong that a nervous irritability of the bladder was established, and the companionship was forced to be discontinued.

Cases may also originate in the gastro-intestinal canal, especially due to overloading of the large intestines, lesions of the rectum and anus, and seat-worms.

The influence of lesions of the external genitals in the production of vesical irritability is well known, and it includes such causes as strictured urethra, retention of smegma and phimosis in the male, and papillomata and other tumors of the urethra and various vaginal lesions in the female.

2. **Increased Frequency of Micturition.**—(*a*) **The Frequency of Quantity.**—"Frequency of urination" is a relative term, as what is frequent for one individual may be normal for another. In any case, then, it is important to determine the normal standard for the individual. Some persons, by reason of the peculiarities of their occupations, are obliged to retain urine for unusually long intervals; hence, they train their bladders to empty themselves at rare intervals. Some are possessed of capacious bladders, and hence may readily get along comfortably by urinating only twice daily. The usual frequency accepted as normal is four to five times daily.

The "frequency of quantity" presents exactly the same clinical indications as polyuria, and the reader is referred to the paragraphs which treat of this subject. The following diagnostic table, quoted from Fenwick, is herewith appended:*

The Frequency of Quantity.

(Much urine, which is passed often.)

Persistent excess.	High s. g.	Sugar.	Diabetes mellitus.
	Low s. g.	No sugar, but extreme thirst.	Diabetes insipidus.
		Albumin with casts, but without pus or residual urine.	Chronic Bright's disease, such as granular kidney, amyloid kidney of advanced scrofulous or syphilitic affections.
		No albumin, but with residual urine.	Back renal pressure from prostatic atony or direct renal irritation of prostatic origin.
Transient excess (usually diurnal).	Low s. g., clear.	(a) Sexual excess or debility (usually without inflammation). Dietetic idiosyncrasy, tea, beer, etc. (b) Hypochondriasis, hysteria, nervousness.	

(*b*) **The Frequency of Irritability.**—It may be stated, in a general way, that frequent urination from irritability of the bladder originates in two classes of cases, (1) in which abnormal composition of the urine is the cause, and (2) those arising from inflammatory and other lesions of the urinary apparatus. Urine of normal composition is the natural stimulus of the bladder to contraction. The influence of unusually dilute urine in promoting abnormal frequency does not seem to be well-recognized clinically, and yet every practitioner has observed cases in which the temporary lowering of the urinary specific gravity has caused frequent emptying of the bladder.

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* Fenwick: "Cardinal Symptoms of Urinary Disease," p. 95.

Of the abnormal urinary constituents capable of producing undue frequency, those worthy of especial note include pus, blood-clots, urates, uric acid, phosphates, and oxalate of calcium crystals. Blood is not included in this list, for there is no evidence to prove that the simple admixture of blood with the urine is capable of disturbing micturition. That hæmaturia is frequently associated with disturbed urination is undoubted; but in nearly all of these cases the undue frequency may be demonstrated to be due to the irritation of blood-clots, or to the lesion giving rise to the hæmorrhage. If the blood is decomposed, it is an entirely different matter, for it then becomes a local irritant, even to the extent of inducing alterations in the bladder epithelium.

In most of the cases in which urinary frequency is associated with lesions of the kidneys, as pyelitis, cystoscopic examination has shown that irritation of the walls of the bladder surrounding the ureteral orifice or orifices is excited by the passage of the pathological urine. It is usually claimed that the urinary irritability in such cases is reflex,—a theory that is untenable in view of the cystoscopic findings. The diagnosis of these cases of urinary irritability, with pyuria, is based upon the principles already stated when treating of the subject of pyuria, the microscope playing an important part in furnishing the necessary data.

The irritability arising from well-marked cases of cystitis is usually readily recognized by the positive symptoms present, including the alkaline urine, the large quantities of pus, the presence of triple phosphates, and the history of a cause capable of producing cystitis.

Other bladder lesions may also produce this symptom, and these include

i. Stone.
ii. Malignant disease.
iii. Papilloma of the bladder.
iv. Ulceration of the bladder.

Lesions of the urethra capable of producing this symptom include:

i. Gonorrhœa.
ii. Stricture.
iii. Prostatitis.
iv. Enlarged prostate.

Fenwick has tabulated the subject of the frequency of irritability as follows:

The Frequency of Irritability.

(Little water, which is passed often.)

1. Without obstruction to the stream.	a. Without pus.	Blood, lithiasis, phosphaturia, oxaluria, dyspepsia.
	b. With pus.	Various irritants in renal pelvis and ureter, *e.g.*, stone, tubercle. Movable kidney. Cystitis of all grades. Catarrhal or tubercular ulceration of the bladder. Hard cancer.
	c. With prostatic threads of pus.	Micturition reflex excited by transient inflammation or congestion of the prostatic M. M., *e.g.*, gout, catarrhal prostatitis. Masturbation, prostatic tubercle and stone.
2. With obstruction to the stream.	Diurnal.	Stone, stricture (6 F gauge), prostatitis, atony (low degree), rare bladder stammerers, vascucular growths of urethra in the female.
	Nocturnal.	Enlarged and congested prostate without much residual urine.
	Diurnal and nocturnal.	Enlarged prostate with residual urine. Cancer of the prostate.

3. **Painful Urination.**—In the majority of cases in which the act of micturition is painful there is a lesion, usually well-marked, of the bladder or urethra. These lesions include cystitis, stone in the bladder, tumors and ulcerations of the bladder, disease of the prostatic urethra, gonorrhœa, urethral stricture, urethral caruncle, and urethral chancre.

A special variety of urinary pain is tenesmus vesicæ or strangury. In this condition, the patient has almost constant desire to urinate, which is done with difficulty, the act being more or less painful. The emptying of the bladder gives but little if any relief. It occurs in all conditions, attended by marked irritation at or about the neck of the bladder. Its presence in dysentery is reflex, from the spasm of the anal sphincter.

4. **Retention of Urine.**—Retention of urine may be obstructive or paralytic, or spasmodic. The obstructive cases are due to stricture of the urethra, enlarged prostate from any cause, and calculus impacted in the urethra. The paralytic cases occur in the course of any spinal cord disorder, in which the cells governing the detrusor fibres are involved, especially, however, in myelitis, locomotor ataxia, and meningitis. The spasmodic cases may be reflex or hysterical. The former originate in inflammatory affections of the urethra, cystitis, dysentery. In this connection it is well to direct attention to the fact that the bladder sometimes overflows as a result of distention, and the idea that the condition is one of incontinence is accepted. This is a very serious diagnostic error, as it leads to erroneous treatment. Such cases can be diagnosed by palpation of the pubic region ; or, failing in that for any reason, the catheter should be passed.

5. **Difficult Urination.**—This may be brought about by any condition leading to weakness of the expulsive force of the bladder, or by in-

creased resistance, *i.e.*, partial obstruction in the lower urinary tract. Its causes then are those of retention, but existing in less degree of severity.

6. **Incontinence of Urine.**—The causes of urinary incontinence may best be presented in the following table:

(a) Spinal cord affections in which the centre for the vesical sphincter is involved.
  i. Locomotor ataxia.
  ii. Ataxic paraplegia.
  iii. Acute and chronic transverse myelitis.
  iv. Disseminated sclerosis.
  v. Spinal meningitis.
  vi. General paralysis of the insane.
  vii. Meningeal and intra-spinal hæmorrhage.
(b) Affections in which unconsciousness or defective cerebration is a prominent feature.
  i. Apoplexy.
  ii. The typhoid state.
  iii. Coma from any cause.
  iv. Sunstroke.
  v. Shock.
  vi. Some cases of insanity and imbecility.
(c) Affections characterized by abnormal or perverted muscular action.
  i. Chorea.
  ii. Epilepsy.
  iii. Tetanus.
(d) Reflex irritation.
  i. Ascarides.
  ii. Phimosis.
(e) Local weakness of the sphincter ani.
  i. From sexual excesses.
  ii. Prostatitis.
(f) Changes in the urine.
  i. Hyperacidity.
  ii. Presence of crystalline sediments.
(g) Conditions throwing unusual strain upon the vesical sphincter.
  i. Vomiting.
  ii. Coughing.

CHAPTER XV.

THE KIDNEYS.

Position of the Kidneys.—The kidneys lie in the posterior portion of the abdominal cavity to either side of the median line, and on a level with the last dorsal and the upper two or three lumbar vertebræ. Their upper extremities are beneath the eleventh and twelfth ribs. The upper extremity of the right kidney is in relation in front with the duodenum and the commencement of the transverse colon; that of the left with the descending colon. The right kidney is in contact above with the liver; the left with the spleen. They are rendered difficult of access from the front by reason of the overlying abdominal viscera, and behind by the surrounding adipose tissue and the large mass of the sacro-spinalis muscle.

The Signs and Symptoms of Renal Disease.—The clinical study of diseases of the kidneys presents certain features not found in the investigation of disorders of other viscera, in that the majority of the symptoms are not referred directly to the kidneys, while others are not experienced by the patient, and are discoverable by special methods of examination. Their investigation, therefore, requires a study of practically the entire body in addition to an examination of the physical condition of the organs themselves and the manner in which they perform their functions.

Among the conditions occurring in other tissues, and which are closely related to renal disorders, may be enumerated

- Cardio-vascular disease.
- Headache.
- Uræmia (*so-called*).
  - Convulsions.
  - Coma.
- Retinitis albuminurica.
- Dropsy.
- Paroxysmal dyspnœa.

To these may be added the symptoms referable directly to the organs themselves:

- The changes found in the urine by chemical and microscopical examination; and
- The physical conditions discoverable by inspection, palpation and percussion.

The headache, uræmic symptoms, retinitis, etc., will be found studied *in extenso* in the sections devoted to the special considerations of headache, uræmia, ocular conditions, etc., respectively. So we proceed at once to a consideration of the physical examinations of the kidneys by inspection, palpation, and percussion.

1. **Inspection.**—Inspection can be of no use in the study of renal diseases excepting in such cases as happen to be attended by unusually great enlargements. In most of these the character of the disease has been determined by other means (palpation) long before the growth becomes evident to inspection. An exception may be made to perinephritis and perinephritic abscess, in which conditions there may be early bulging, associated with redness and œdema of the overlying skin. Sometimes, too, in these cases there will be associated fluctuation, and the constitutional and local symptoms of suppuration, including chills, fever, and pain.

2. **Palpation.**—The value of palpation in the diagnosis of renal enlargements and misplacements has been greatly underrated. Notwithstanding the views of many physicians, the kidneys may be studied by palpation, if care be taken in practising it. Bimanual manipulation is necessary. The patient should lie on the side opposite to the kidney under examination, with the thighs flexed on the abdomen and the muscles thoroughly relaxed. One hand of the examiner is next placed upon the back below the last rib and outside the lumbar muscles. The other hand is placed on the abdomen in front, and opposite the other hand. Simultaneous pressure and manipulation are now made with the hands, and the kidney sought for is readily brought under the touch of the examining fingers. Care should be taken that the stomach and colon are empty.

Musser suggests, as a most excellent position for palpating the kidneys, standing and leaning over the back of a chair, with the hands resting upon the seat of the same, supporting the trunk. In this position the abdominal muscles are well-relaxed, and the kidneys fall forward by gravity, if their ligaments are at all relaxed.

Palpation enables us to discover the presence of

(a) Renal displacements.
(b) Renal enlargements.

(*a*) **Renal Displacements ; Floating Kidney ; Wandering Kidney ; Nephroptosis.**—This is a condition occurring with remarkable frequency. If physicians were as active as the gynæcologists with their "educated touch," it would probably be found to be as common as the much-talked-of prolapsus uteri. There can be no question that many of the obscure symptoms of invalided women are due to it and associated displacement of abdominal viscera ; indeed, I might also say that, in all probability, careful examination would show that pelvic displacements are not uncommonly associated with this condition of the kidney.

Nephroptosis may be either congenital or acquired. The congenital condition is the more rare of the two, and is due to the formation of a true mesentery for the kidney. The acquired variety occurs with especial frequency in women with relaxed abdominal walls, and who have had several pregnancies. A movable kidney is readily discernible by palpation as a movable, slippery tumor. It presents the characteristic kidney-shape. The right is affected more frequently than the left. The tumor may be found in any portion of the abdominal cavity, and even in the pelvis. The position normally occupied by that kidney is found, by inspection, to be less full, and, on palpation, to be less resistant than the opposite side. Manipulation readily restores the organ to its normal position, causing the tumor to disappear. Pressure upon it gives rise to a peculiar pain or sickening sensation, which is more marked during the menstrual periods. In some cases, it is possible to feel the pulsation of the renal artery in the hilus of the kidney.

(*b*) **Renal Enlargements.**—These include :

i. Renal abscess.
ii. Hæmorrhagic infarcts.
iii. Malignant tumors (particularly sarcoma).
iv. Renal hypertrophy.
v. Pyonephrosis.
vi. Hydronephrosis.
vii. Cysts.
  i. Simple cysts.
  ii. Dermoid cysts.
  iii. Hydatid cysts.

Let us first consider the characteristics by which we recognize kidney enlargements in general ; after which it will be in order to study the characteristics of individual cases.

Renal tumors in general start well forwards in the lumbar region, extending thence upwards into the hypochondrium, and backwards towards the spinal column, forwards to the umbilicus, and downwards to the iliac crest. A hollow can usually be felt between the upper margin of the tumor and the costal margin, a most important point, as it serves to differentiate tumors of the left kidney from those of the spleen. They are always situated behind the colon, another important point for differentiation, as splenic tumors are always in front of the colon. When the colon is well distended there is no difficulty in its recognition. Sometimes, however, it extends across the renal tumor as a thickened cord, thus giving, on palpation, the impression that there are two tumors instead of one. If any other than the above-mentioned data are needed to differentiate renal from splenic tumors, we have the shape to guide us, those of the kidney presenting a rounded anterior margin, those of the spleen having a sharp-notched edge. Splenic tumors rarely cause enlargements behind, but push forwards. While respiratory mobility is to be regarded as contraindicating the presence of renal tumor as a rule, it must be remembered that these have some slight motion during respiration.

The individual enlargements of the kidney now come up for consideration.

i. **Abscess.**—Abscess of the kidney presents a rounded, possibly fluctuating enlargement, associated with some fullness of the loins. The affected kidney is tender; indeed, may be painful. Microscopical examination usually discovers the presence of pus and blood in the urine. The constitutional symptoms of suppuration are usually present. One of the prominent causes of renal abscess being tuberculosis, the urine should be examined for the presence of tubercle bacilli.

ii. **Hæmorrhagic infarcts** are of sudden onset, and are especially apt to occur in association with a septic disorder, especially malignant endocarditis.

iii. **Malignant tumors** present a nodular feel. They are accompanied by severe pain, and not infrequently by hæmaturia, which may be produced by a very slight traumatism.

iv. **Renal Hypertrophy.**

v. **Pyonephrosis** presents a tense, smooth, globular tumor, with fluctuation. Tenderness is absent. Pus is discharged copiously in the urine from time to time, such discharges being attended by a corresponding reduction in the size of the tumor.

vi. **Hydronephrosis** is due to obstruction of the corresponding ureter, damming the urine back in the pelvis of the kidney, virtually transforming that organ into a cyst. The pressure thus generated leads to glandular atrophy. The tumor produced presents the same physical characteristics as pyonephrosis. There is absence of fever, however. The tumor subsides rapidly, sometimes in association with an unusually free passage of urine, due to the relief of the obstruction, to reappear on its renewal. In doubtful cases the tumor may be aspirated, when there will be drawn a turbid fluid containing epithelial cells. The intermittency of some cases of hydronephrosis may lead to the mistaken diagnosis of wandering kidney.

3. **Percussion.**—As a means of investigating the physical condition of the kidneys, percussion is not, in view of the anatomical conditions prevailing, apt to give satisfactory results. The kidneys lie upon the sacrolumbalis muscles, which have a large bulk, so that percussion can with difficulty differentiate what is muscle and what is dullness of the organs. It is, however, of great value in determining the relationship of a distended colon to a tumor under examination,—a very important point, as already stated. Percussion is favored by having the patient lie face downwards, with pillows under the abdomen, thus throwing the lumbar region well upwards. A result is only possible when forcible blows are delivered, and plexor and pleximeter are employed. Better results will be obtainable if, instead of endeavoring to delimit the kidneys, the examiner direct his entire attention

to determining the angle between the right kidney and liver dullness on one side and that of the splenic and left kidney dullness on the other.

Pain in Diseases of the Urinary Apparatus.—Pain is not a prominent feature in the majority of cases of renal disease. This view is in direct contradiction to ideas prevalent among the laity, who are inclined to attribute pain in the back to disease of these organs. Backache is, in the majority of cases, dependent upon lumbago. Still, there are numerous exceptions to this statement. Sometimes there occurs a dull, aching pain, aggravated on movement, usually bilateral, and associated with some sense of weight and oppression, as symptomatic of renal disease. In the majority of cases in which it occurs its presence is overshadowed by other phenomena, so that it ofttimes fails of recognition. The general rule governing the production of pain from kidney disease may be stated as follows: Lesions which cause rapid distention of the capsules of the organs, or which irritate the lining membrane of the renal pelves, produce pain. It is, then, the situation and mode of growth of the lesion, rather than the character of the latter, that is the pain-producing factor. At the same time, it must be remembered that certain conditions are far more likely to produce pain than others. These include calculus, malignant disease, and tuberculosis. But if they progress slowly, so as not to suddenly distend the capsule, it is possible for the secreting structures of the kidneys to be entirely disorganized without any pain whatever.

The diseases included under the generic term "Bright's disease" are not attended by pain because the pathological changes cause no capsular distention. Acute nephritis, by reason of the rapid swelling of the organs affected, is usually painful.

Unilateral renal pain is sometimes observed. It may be described as situated in the back over the region of the kidney, or deep in the abdomen to one side of the median line, and just below the level of the umbilicus. In the vast majority of instances it indicates the side affected. But exceptions have been observed in which the pain is referred to the sound kidney. Such exceptions are, however, not so frequent as they appear to be; for it has been demonstrated that the so-called sound kidney was the seat of a lesion capable of exciting pain.

The usual pain of malignant disease, when present, is constant. When the lesion has involved the pelvis, that fact is indicated by the radiation of the pain along the corresponding ureter to the bladder, and, it may be, to the thigh or sole of the foot.

Paroxysmal pains attend the passage of a calculus from the pelvis of a kidney along the ureter to the bladder,—renal colic. The pain is referred to a line extending from the kidney to the side, and forwards and downwards to the region of the bladder. As a rule, the suffering is atrocious. It seems to stand in inverse ratio to the capacity of the ureters. It is due not only to the actual irritation of the passage of the calculus, but also to the damming back of the urine. In severe cases the pain may extend into the testicle and end of the penis. Blood is found in the urine as a rule. Attacks simulating renal colic may be produced by obstruction of the ureter, by a blood-clot, a portion of detached tumor, or hydatid cyst. Renal calculus is sometimes accompanied by a steady pain, situated on either side of the abdomen, opposite the brim of the pelvis.

Pains simulating those of renal colic sometimes occur in gout, and are due to the irritation of particles of uric acid passing along the ureters. It is said, also, that they may result from the irritation of pus or blood in the urine.

A rare painful condition of the kidney is the so-called **neuralgia of the kidneys.** It is said to be produced by malaria, gout, lead-poisoning, and anæmia. Such cases are often reported by physicians, but, as a matter of fact, the majority are examples of diagnostic errors. As skill in investigating disease increases, they will be reported with decreasing frequency.

The only certain renal neuralgia is the *nephritic crises* of locomotor ataxia. The pains resemble those of calculus; but hæmaturia is absent, and the patient presents the absent knee-jerks, the Argyll-Robertson pupils, and the other characteristic symptoms of tabes.

Synopsis of the Symptomatology and Clinical Course of the More Common Diseases of the Kidneys.

1. Congestion of the Kidneys.

Congestion of the kidneys may be *active* or *passive*.

(*a*) **Active congestion of the kidneys** can be separated, clinically, from acute nephritis only with the greatest difficulty, because it constitutes the initial stage of that disease. It is therefore hard to determine where one ends and the other begins. Etiological factors afford an important diagnostic clue, especially when considered with reference to associated symptoms and conditions. The causes include the irritation of poisons introduced from without, as cubebs, copaiba, cantharides, turpentine and quinine, and the elimination of toxic substances formed within the body, as from the disturbed metabolism of pregnancy, and in many of the infectious diseases. It may also result from irritation of the lower urinary tract.

The symptoms in many instances are of the most indefinite character, including mainly the urinary changes. Sometimes, however, the patient complains of discomfort in the back. The urine is diminished, though rarely to an alarming extent, as in acute nephritis. It always contains more or less blood, though usually the quantity of that substance is small. It is albuminous, though usually to but slight degree. Casts are generally discoverable, and include the hyaline, granular and blood varieties. Dropsy is never present. We rely upon the last-named fact to differentiate it from acute nephritis. It will be seen from the above statements that active renal congestion is never a primary affection, excepting in those instances in which it constitutes the initial stage of acute nephritis.

(*b*) **Passive Congestion of the Kidneys.**—This, likewise, is a secondary condition, being the result of diseases which impede the return flow of venous blood, and which include especially heart diseases with ruptured compensation, pulmonary emphysema, cirrhosis of the liver, thrombosis of the renal veins, and tumors obstructing the ascending vena cava. Clinically, the presence of passive congestion of the kidneys is determined by great diminution in the daily quantity of urine in conjunction with one of the causes above named. Usually, the urine is dark in color and of high specific gravity. It may or may not be albuminous. Blood may be discovered in some cases. It is not uncommon to find the liver enlarged from passive congestion. From actual organic disease of the kidneys associated with heart disease, it is to be differentiated by the relatively small amount of albumin, the high specific gravity, and the presence of hyaline and granular casts only.

2. Acute Nephritis; Acute Bright's Disease.

The most frequently-observed cause of acute nephritis is exposure to cold, especially in those addicted to excessive use of alcohol. Of late there has been a growing tendency to underestimate the importance of this cause by persons who claim that the relationship is a theoretical one only. Such skepticism has actual experience to show its fallacies, for in most of the cases of acute nephritis the history of exposure is undoubted. So far as we can determine, alcohol of itself is unable to produce acute nephritis. The next cause in order of frequency is the poisons of the acute infectious fevers, notably scarlatina, typhoid fever and diphtheria. The persistent albuminurias of pregnancy are almost certainly due to acute nephritis. Some few cases follow extensive lesions of the skin.

The initial symptoms include such constitutional disturbances as rigor succeeded by fever, in which, however, the temperature rarely goes above 103° F., pain in the back, nausea and vomiting. The urinary secretion is early affected, as exhibited by its diminished quantity, high specific gravity, smoky color, and the large proportion of albumin which it contains. Dropsy appears early, and may be the first symptom to which the patient attaches more than a passing importance. Usually the patient is more or less anæmic. The *dropsy* first asserts itself about the eyelids, later spreading over the entire body. Not infrequently the serous cavities, as the pleuræ and the peritoneum, are extensively involved. Physical examinations of the patient should be repeated regularly, for it may happen that the effusions in the serous cavities may be disproportionately great, and otherwise escape observation. The *urine*, besides being highly albuminous, is found microscopically to contain blood-cells, urinary epithelium, and hyaline, blood and epithelial casts. The daily quantity of urea excreted is much below the normal. *Cardiac complications* are not common. Exceptionally, acute dilatation may set in and lead to a fatal issue. As a rule, vascular tension is increased; in which case, the aortic second sound will be found to be accentuated. If the case pursues an unfavorable course, the urinary excretion becomes completely suppressed, and the complexus of symptoms which we commonly designate as uræmia appears, and usually carries off the patient. From the complete clinical picture of acute nephritis as above presented, variations are observed in practice. Thus, there may be a slight œdema leading to the discovery of the urinary changes; or it may even happen that the latter alone may be present. While acute nephritis is always a serious disease, the majority of cases recover. The prognosis of those following upon exposure to cold is generally regarded as more favorable than the post-scarlatinal cases. In the favorable cases, too much must not be counted upon the early disappearance of the albumin; and, again, the persistence of albuminuria for several months need not be regarded as a serious matter, but simply as a warning to exercise close supervision over the patient's mode of life.

3. Chronic Parenchymatous Nephritis. Chronic Diffuse or Tubal Nephritis; Large White Kidney.

This disease presents some variety in its clinical course. In one class of cases the patient presents a number of vague symptoms of sufficient severity to warn him that he is not in good health, and the real state of affairs is discovered only when the urine has been examined. Usually, however, anæmia is noted early, and is progressive in character. After the disease has continued for some time, œdema appears, and, as is usually the case in renal affections, first involves the tissues about the eyelids. Like the anæmia, it is progressive, and finally spreads over the entire body. The serous cavities are frequently invaded, though not usually until an advanced stage of the disease. Throughout the course of the disease the patient exhibits a tendency to digestive and respiratory disturbances. The latter, which are the more frequently encountered, include bronchitis, catarrhal pneumonia, œdema of the lungs, and pleurisy. Serous membrane inflammations are occasionally observed. While all the symptoms above mentioned are highly suggestive, it is upon the urinary condition that we must base our diagnosis. The daily quantity of urine is diminished according to the progress of the disease. At first its specific gravity is normal; later, it becomes low. Albumin is always present in large quantity,—larger, indeed, than in any other chronic renal disease. The urea excretion is lessened. Microscopically, tube-casts of all varieties are observed, together with red-blood cells, leucocytes, and degenerated renal epithelium. Cardiovascular changes ultimately ensue, and include hypertrophy of the heart, increased arterial pressure, and general thickening of the arteries. During the later months of the disease albuminuric retinitis is not uncommon, and is always to be regarded as an unfavorable sign. Ultimately uræmia appears, generally taking the form of coma, though sometimes the convulsive type is observed. The prognosis of chronic parenchymatous nephritis is highly unfavorable. Some few cases recover, but they do not include cases which have continued longer than a year or eighteen months. The outlook seems to be somewhat better in children.

4. Chronic Interstitial Nephritis; Gouty Kidney; Granular Kidney; Contracted Kidney.

In the affection bearing the above designations we have one of the most common causes of ill-health in persons who have passed the age of fifty years. Treacherous in its onset, its presence is ofttimes unsuspected until it has become well-advanced, and one of its numerous complications has asserted itself and made medical attention a necessity. The causes leading up to interstitial nephritis are many and varied. It is in exceptional cases only that any one of them is alone productive of the case in hand. Predisposition is in many instances a powerful factor, as is evidenced by the family tendency to the disease exhibited by certain cases. The heredity may be direct, in that the disease transmitted thus is chronic interstitial nephritis, or it may be by the simple inheritance of the gouty constitution, itself an important cause of the disease under consideration. The determining causes involve numerous violations of the laws of health as relating to eating, drinking, exercise, rest and recreation. Important as are alcoholic excesses in the production of this, as of other kidney diseases, I believe that excesses in eating, especially in persons who lead a sedentary life, are much more so. Nitrogenous and highly-seasoned food is especially hurtful. Some cases of interstitial nephritis exhibit a generalized arterio-sclerosis as their primary manifestation. In them the usual cause is found in a life of great mental and physical strain and activity, ofttimes combined with worry.

No disease is capable of as great a variety of clinical manifestations or of as many complications as is chronic interstitial nephritis. The reason for this is clear, when one considers the widespread vascular changes with which it is practically always associated. At the same time, the lesions progress so slowly that the system accommodates itself to the new state of affairs, and thus subjective evidence of ill-health is not manifested until a late stage of the disease. The essential features of chronic interstitial nephritis may be tabulated as follows:

(a) Urine of low specific gravity, usually increased in quantity, generally containing a small percentage of albumin and a few hyaline casts.

(b) The cardio-vascular changes, as exhibited by hypertrophy of the left ventricle, general arterio-sclerosis, and increased intravascular pressure.

Subordinated to these, but commonly present, though not essential to the diagnosis, are:

(a) Gastro-intestinal disturbances, as exhibited by defective digestion.

(b) General weakness, manifested by progressive incapability for mental and physical work.

Symptoms of the advanced stages include

(a) Œdema of the eyelids, and sometimes of the ankles, though at no time of great amount.

(b) Retinitis albuminurica.

(c) Hæmorrhages, as epistaxis, cerebral hæmorrhage, hæmaturia.

The Urine.—In the early stages of chronic interstitial nephritis the urine is increased in quantity, as a rule. Sometimes this polyuria is well marked, as much as 100 or more ounces of urine being passed daily. This necessitates frequent rising to urinate during the night, and is attended by more or less thirst. The urine is light-yellow in color, and of specific gravity ranging from 1005 to 1012. This persistent low specific gravity, taken in connection with the cardio-vascular changes, is sufficient to establish a diagnosis in the early stages. In the majority of cases, albumin is present in small amount. When it is alleged to be absent, repeated examinations will discover its occasional presence, especially in urine passed towards the close of the day. Casts are always few in number; indeed, it may only be after the examination of a number of slides that they can be discovered. They are always of the hyaline or granular type.

The Cardio-Vascular Changes.—Very early in all cases the arteries undergo thickening, and at the same time the intravascular tension becomes increased. This has its effect on the heart, by reason of which that organ undergoes hypertrophy in order to accomplish the extra work demanded of it. With the advent of the fully-developed disease, the heart, no longer able to maintain its extra work, dilates, and we have evidences of failing compensation, dyspnœa, gallop rhythm, etc.

Gastro-Intestinal Disturbances.—These consist for the most part of loss of appetite and discomfort after eating. Occasionally they are more pronounced, and may constitute the first phenomena to drive the patient to his physician for thorough examination. The prominent features in the severe cases include, especially, uncontrollable vomiting and diarrhœa. In two cases under my care, severe intestinal hæmorrhage,—one of them fatal,—was the initial symptom.

General Weakness.—By reason of the extensive vascular changes, the nutrition of all tissues must be impaired, and this lessens the patient's capabilities for work, both mental and physical. Exceptionally, no trouble in this direction is experienced, the patient maintaining his usual pace almost to the closing hours of his life.

Œdema is less prominent in chronic interstitial nephritis than in any of the affections classed under the general term of Bright's disease. As already stated, it is confined to slight puffiness about the eyelids and swelling about the ankles. When at all prominent, it is due to the associated dilatation of the heart.

Retinitis albuminurica does not appear until the case nears its fatal termination. As a rule, this symptom means that the patient will die within six months, and it is very unusual for the result to be postponed more than a year after its discovery. What is remarkable, every oculist can tell of cases in which the first suggestion of the correct diagnosis was made by the ophthalmoscope.

Hæmorrhages.—Cerebral hæmorrhage is a common means to the fatal termination of interstitial nephritis. The vascular degeneration, the cardiac hypertrophy and the increased pulse-tension make it so. Hæmorrhages in other portions of the body are probably as frequent, but, owing to their less serious character, do not attract the attention their importance merits. In the case of hæmoptysis from this cause, the great frequency with which tuberculosis is the cause of this symptom leads to the failure to discover the nephritis.

Some unusual as well as remarkable respiratory disturbances appear during the course of some cases. They take the form, usually, of asthma, which continues for a few days, and then disappears as suddenly as it came.

5. Amyloid or Lardaceous Kidney.

Amyloid degeneration of the kidneys never occurs excepting as the result of long-standing suppuration, syphilis or bone disease, and is almost invariably associated with similar pathological changes in other portions of the body, especially, however, in liver, spleen or intestines. Its diagnosis rests upon the discovery of these associated conditions taken in connection with the history of the case, for there is nothing in either symptomatology or urinary changes to separate it from the various forms of Bright's disease. Because of the great polyuria in the early stages, it may be mistaken for interstitial nephritis, but from which it differs in that the cardio-vascular changes are always absent. Albumin is present in greater amount than in contracted kidney; but when the quantity of urine is large, its percentage is apt to be very low. Dropsy is a more prominent feature in the later stages, when the urine becomes scanty, than in other renal affections. The discovery of a relatively large proportion of globulin, as compared with serum albumin, is regarded as suggestive of amyloid disease. But, afte all that has been said from the clinical side of the question, we must rely for our diagnosis on the history of the case and the associated signs and symptoms.

6. Pyelitis, Pyelonephritis and Hydronephrosis.

Pyelitis is an inflammation of the pelvis of the kidney. When the inflammation spreads to the renal structure, we call it a pyelo-nephritis; and when, by reason of obstruction of a ureter, pus is dammed up in the pelvis, we have the condition known as pyonephrosis. Inasmuch as two of these conditions are phases of pyelitis, we shall use the latter term generically in the following description. Many cases originate in an extension of purulent inflammation from the lower portion of the urinary tract, as cystitis, gonorrhœa, calculus, etc. Such inflammations are especially liable to involve the renal pelves in paralytic retention of urine attendant upon myelitis and other diseases of the spinal cord. Unclean catheterization is sometimes the cause. Sometimes the inflammation starts primarily in the renal pelvis as the result of infection carried thence by the blood, or renal secretion, as in typhoid fever, tuberculosis, pneumonia, and scarlatina. Traumatism may afford the explanation in cases of renal calculus and after severe blows upon the back. It may occur during the course of ulcerative endocarditis, and is then due to infection by a septic embolus. Irritant poisons, as cantharides, turpentine and copaiba, have been assigned as causes; but this seems problematical, unless the irritation is associated with an infection.

The symptoms include the constitutional phenomena of suppuration, the local subjective manifestations of a suppurative inflammation, and the urinary changes. The constitutional symptoms vary in severity according to the extent of the lesion. In the initial stage of all cases we have rigors succeeded by fever, these phenomena being the more severe when the renal structure itself is involved. This fever pursues the ordinary septic type.

When the renal pelvis alone is involved, other constitutional disturbances are not prominent; but when the case has become one of pyelonephritis, more or less rapid emaciation sets in. At the same time the patient assumes a sallow or cachectic appearance. When this condition has continued for a long time, the typhoid state supervenes, with nausea, vomiting, and cramps, and finally death by exhaustion or uræmia. *Locally*, the patient complains of pain in the back, with sensitiveness on deep pressure over the region of the kidneys. The *urine* is of acid reaction and contains pus. In simple pyelitis, any albumin present is due almost entirely to the purulent admixture. Sometimes, owing to obstruction of the ureter, pus disappears entirely from the urine for a variable length of time. When the disease invades the renal structure proper, albuminuria becomes a more prominent feature, and tube-casts are common. In all cases the sediment contains renal epithelium. The diagnosis of pyelitis is, as a rule, not a difficult matter. The main problem is the differentiation of the tubercular from the calculous cases. Ofttimes this question can only be decided by a painstaking search for the tubercle bacillus. It has been suggested that the X-ray will enable us to detect the presence of a renal calculus.

7. Hydronephrosis.

The physical signs of hydronephrosis have been presented when speaking of palpation in the diagnosis of renal diseases (p. 570). It only remains at this time to speak of the causes leading up to the condition. These include all agencies producing ureteral obstruction, of which the most frequently observed are impacted calculus, cicatricial stenosis of a ureter, the pressure of a tumor upon the ureter, especially in uterine carcinoma, fibroids, kinking of a ureter from movable kidney, and by traction of adhesions succeeding peritonitis. Congenital cases dependent upon imperforate urethra have been reported. The enlargement of the kidneys in these cases may be so great as to be a serious cause of dystocia.

8. Nephrolithiasis.

The symptoms from renal calculus present the greatest possible variations. For years the stone may remain in the renal pelvis, giving rise to little or no discomfort. Sometimes a pyelitis is excited. The most characteristic phenomena, however, are those attendant upon the passage of the stone down along the ureter, producing the symptoms known as *renal colic*. The paroxysm usually sets in suddenly with severe pain, which extends from the kidney on one side along the ureter to the bladder, and may even be experienced in the testicle and side of the thigh on the corresponding side. Exceptionally, the pain extends upwards as well, and appears in the chest. In some cases the constitutional disturbance is profound, as shown by nausea and vomiting, coldness of the surface, weak pulse, and intense prostration. The duration of the paroxysm is indefinite, in that it may be over in little more than an hour, or it may persist for several days. It is not unusual to find urination frequent, and sometimes it is painful. The diagnosis of renal calculus cannot be regarded as mathematically certain, unless, in association with the pains, there is hæmaturia. Obstructive suppression of urine may result, the calculus involving the ureter of a sound kidney, the other one being extensively diseased. Death from uræmia may result in these cases. Following an attack of renal colic, the patient usually complains of soreness and aching in the affected side. When a calculus lying in the renal pelvis excites symptoms, they include pain in the back, usually amounting to nothing more than soreness, a fitful hæmaturia, and the usual phenomena of pyelitis.

9. Perinephritis; Perinephric Abscess.

Perinephritis is usually the result of the extension of a suppurative lesion of the kidneys, as pyonephrosis or renal tuberculosis. Cases may also originate in infection from abscesses in other portions of the body, as in appendicitis, pelvic suppuration, pelvic abscesses, disease of the vertebræ, affections of the rectum, abscesses of the liver and spleen, and in the course or as one of the sequelæ of certain infectious diseases, as typhoid fever, scarlatina and variola. It may originate in direct traumatism. Its symptoms include the ordinary constitutional manifestations of suppuration, as rigors, remittent fever, profuse sweating, and prostration. Locally, the patient complains of severe pain in the back, forcing him to flex the knee and thigh on the affected side for relief. Finally a swelling appears in the lumbar region, the cutaneous surface over which is unduly red, and is doughy to palpation. The skin becomes œdematous, and finally fluctuation is plainly discernible. The urine does not give any evidence of renal disease, excepting in those cases in which the abscess cavity communicates with the renal pelvis.

10. Cysts of the Kidneys.

Quite a variety of cystic formations may invade the kidneys, as follows: (*a*) In which *numerous small cysts* are engrafted upon granular kidney, and are due to obstruction of single uriniferous tubules. (*b*) *Single large cysts*, usually occupying one or the other extremity of the kidney, generally the upper. These cysts are commonly believed to be due to tubular obstruction or external violence. (*c*) *General cystic disease*, in which the renal substance is replaced by a large bunch of cystic formations. This latter variety may be either congenital or acquired. In the latter case, the lesion is nearly always bilateral. The affected organs attain an enormous size. In all the varieties of renal cysts it is important to recognize that there is always considerable normal kidney tissue remaining, and this fact impresses itself on the clinical course of the disease.

A variety of types, according to their clinical manifestations, is also recognizable: (*a*) In which the lesion produces no symptoms whatever during life, and is discovered only at the autopsy. (*b*) In which the disease pursues a latent course for a number of years, when suddenly a fatal uræmia appears. (*c*) In which a definite group of symptoms sufficiently characteristic to establish a diagnosis, appears.

The symptoms in the class last-named include progressive loss of strength and emaciation, with the excretion of large quantities of urine of low specific gravity. Physical examination discovers bilateral tumors in the region of the kidneys. Death usually ensues from uræmia. The latter may set in suddenly, and be characterized by convulsions, delirium and coma; or it may follow the latent type, in which case the symptoms include nausea, cramps, muscular twitchings, subnormal temperature, and contracted pupils. The discovery of a bilateral renal tumor constitutes a very important diagnostic symptom.

11. Hydatid Cysts of the Kidney.

Hydatid cysts of the kidney are of rare occurrence. They can be diagnosed only when the tumor has communication with the renal pelvis, and portions of the cyst or scolices are discharged in the urine. The escape of the latter will, of course, be attended by the ordinary phenomena of renal colic. Palpation usually discovers a renal tumor. The presence of the characteristic hydatid thrill is not reliable; pyelitis and cystitis are not uncommon complications.

12. Renal Tuberculosis.

Renal tuberculosis may exist (*a*) as part of the manifestations of a general infection; or (*b*) as one of the phases—usually the initial one—of a general urinary tuberculosis. The latter is by far more common than is usually supposed. The symptoms are those of pyelitis or pyelo-nephritis. The diagnosis rests upon the discovery of tubercular lesions in other portions of the body, or upon the presence of the tubercle bacilli in the urine.

13. Malignant Disease of the Kidneys.

The malignant diseases which may involve the kidneys include sarcomata and carcinomata, the former being the more frequent. They may occur as a primary lesion, or as secondary to similar malignant processes elsewhere. Renal sarcoma is especially common in childhood. The tumor may attain an enormous size. The first symptom of malignant disease of the kidneys is usually hæmaturia, which may take place spontaneously or in response to apparently insignificant traumatism. Excepting in those cases in which the blood escapes rapidly, the urine presents the characteristics usually observed in renal hæmorrhage. Pain is ofttimes present, but is so frequently absent that it cannot be regarded as of much importance from a diagnostic standpoint. The constitutional symptoms are generally present, though they may be delayed until late in the course of the disease. The diagnosis cannot be established, of course, until palpation has discovered a tumor in the region of the kidneys.

14. Diabetes Mellitus.

For the present we are obliged to regard the term "diabetes" as a generic one, applied to a group of conditions having in common glycosuria, polyuria, thirst and emaciation. Our knowledge concerning the pathology of this interesting symptom-group is but limited. We have thus far reached the conclusion that it may be brought about by causes relating to the pancreas, to faulty diet, and to nervous agencies.

It is upon the urinary changes that the diagnosis is based. The patient passes several times the normal quantity of urine in the course of the twenty-four hours. Its specific gravity is high, even to 1040 to 1050; it contains sugar—in extreme cases as much as 12 per cent. Too much reliance must not be placed upon the high specific gravity, as occasionally sugar is found in urines of abnormally low gravity. Thirst is extreme, impelling the patient to drink at very short intervals. The patient's appetite is good, as a rule; notwithstanding which, he loses flesh.

Aside from the above symptoms, diabetes may exhibit numerous others which are to be regarded in the light of complications. Skin symptoms are especially frequent in this connection. They include the formation of pustules or boils, symmetrical erythema of the face, eczema, and xanthoma.

Closely related to the skin manifestations is the diabetic gangrene. This may appear as small areas of necrosis arranged about the orifices of the sweat-glands. It may be attended by bullæ, the gangrene appearing in patches, as described by Kaposi (*Gangrena bullosa serpiginosa*), and is probably dependent upon neuritis. The most frequently-encountered variety is due to vascular disease, occurs more frequently among men, especially after middle life, and renders amputation necessary.

The nervous complications are quite numerous, and are often the subjects of diagnostic errors. Neuralgic affections are common. As a rule, they are bilateral. Especially important in its relation to diabetes is sciatica. Multiple neuritis occurring in the course of diabetes is very liable to be mistaken by the uninitiated for locomotor ataxia. It is stated by Gruber that diabetic neuritis may occasionally simulate tabes dorsalis so closely as to make a clinical differentiation an impossibility.

Of the ocular complications, cataract is the best known. Of recent years, attention has been directed to the fundus of the eye, as several varieties of retinitis have been observed. Amblyopia similar to that produced by excessive indulgence in tobacco has been observed, and it has been suggested that diabetics may be more than ordinarily susceptible to the deleterious effect of tobacco on the eyes.

Many cases of diabetes terminate in coma. Previous to this accident the urine diminishes in quantity, and is found to contain acetone and diacetic acid. Respiration becomes accelerated; the patient may exhibit unusual weakness; and then the serious symptoms appear. These are manifested as restlessness, delirium, epigastric pain, and rapid and feeble pulse. Then drowsiness sets in, and deepens into coma.

CHAPTER XVI.

THE BLOOD.

THE functions of the blood being of great physiological importance, it is natural to assume that an examination of its structure and chemical composition should aid us greatly in clinical investigations. Unfortunately, hæmatology is such a new science that the examination technique can hardly be said to be perfected, and there are many observable facts to which the proper interpretations have not been made. Notwithstanding the present uncertainty, there is much information of positive value obtainable by systematic examination of blood-specimens. As in the case of the urine, there are very few diseases in which the condition of the blood is sufficient of itself to establish the diagnosis. If we were to limit hæmatology in its clinical relations to pathognomonic symptoms, we would find its study of but little practical value. It is to be regarded as furthermore useful in the discovery of conditions which dissolve doubts in the differentiation of certain similar clinical conditions; in the discovery of data which suggest the existence of a disorder hitherto unsuspected; and in the negativing of the possibility of conditions which the associated symptoms had led us to suspect as being present.

An examination of the blood includes attention to the following:

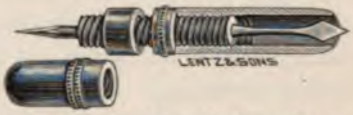
1. Determination of the specific gravity.
2. Determination of the alkalinity of the blood.
3. Estimation of the percentage of hæmoglobin.
4. Determination of the coagulability of the blood.
5. Determination of the number of red corpuscles per cubic millimetre.
6. Determination of the number of white corpuscles per cubic millimetre.
7. The examination of dried and stained blood-specimens, by which may be determined the presence of abnormalities
    a. In the red cells, including
        i. Abnormalities of shape.
        ii. Abnormalities in size.
        iii. Abnormalities of structure.
    b. In the white cells, including
        i. Departures from the normal of the relative proportions of the several varieties of leucocytes.

ii. Abnormalities of shape, size and structure of leucocytes.

8. Examinations for parasites.

For the purpose of obtaining a specimen for examination, no instrument is better than the especially-devised lancet, a figure of which is here appended. The part selected for puncture should be the palmar surface of the terminal phalanx of a finger or the lobe of one ear. It should first be thoroughly cleansed with a wet cloth, and then dried with brisk friction. This tends to produce an increased determination of blood to the part, and promotes free bleeding. Antiseptic precautions do not seem to be necessary, although, when operating on nervous subjects, their adoption is probably wise. The first few drops which escape should be wiped away. No attempt whatever should be made to promote bleeding by pressure, as blood obtained in this manner must contain an abnormally large percentage of blood-serum, and cannot, therefore, represent the general corpuscular richness of the blood.

FIG. 147.



Lancet for obtaining blood-specimen.

1. **Determination of the Specific Gravity of the Blood.**—The specific gravity of the blood ranges from 1045 to 1075. It is higher in men than in women, and at birth than at subsequent periods of life. During adult life the maximum is observed between the ages of 35 and 45 years. It is lessened by hunger, during pregnancy, and by exercise. The clinical importance of determining the specific gravity of the blood lies in the fact that it varies directly with the hæmoglobin percentage. Inasmuch as the latter requires for its determination an instrument involving some expense, the specific gravity may be utilized as an indirect means for the estimation of the hæmoglobin.

The best method for obtaining the specific gravity of the blood is that of Hammerschlag. A mixture of equal parts of chloroform and benzol is made in a urinometer glass. The chloroform is heavier than blood, and benzol is lighter. A drop of blood is placed in the mixture, care being taken that it does not adhere to the walls of the glass. It will not incorporate itself with the mixture, but will rise or fall according to its relative density with the fluid. The specific gravity of the mixture may be increased or diminished by adding chloroform or benzol, respectively, until a point is reached when the blood-drop remains suspended. Then the specific gravity of the mixture is taken with an accurate urinometer, and the result represents the specific gravity of the blood. After using, the fluid may be filtered and bottled until another examination is necessary. Hammerschlag gives the following tables as aiding in determining the hæmoglobin percentage from the specific gravity :

Specific Gravity.	Hæmoglobin.
1033–1035	25–30 per cent.
1035–1038	30–35 "
1038–1040	35–40 "
1040–1045	40–45 "
1045–1048	45–55 "
1048–1050	55–65 "
1050–1053	65–70 "
1053–1055	70–75 "
1055–1057	75–85 "
1057–1060	85–90 "

The specific gravity is not applicable for the determination of the hæmoglobin percentage in cases of dropsy, because in that condition the specific gravity of the blood-plasma itself is altered, and the quantity of hæmoglobin contained is not the only factor causing a departure from the normal standard.

2. **Determination of the Alkalinity of the Blood.**—As yet, the determination of the alkalinity of the blood is not of much clinical importance. Normally, the reaction of the blood is alkaline, owing to the presence of disodic phosphate and bicarbonate of soda. The degree of alkalinity may vary under physiological as under pathological conditions. No practical method has been devised for stating the normal standard.

The alkalinity diminishes when blood is withdrawn from the body,—the more so, as the blood is permitted to stand the longer. It is also found reduced in fever, uræmia, carbonic oxide and phosphorus poisoning, organic diseases of the liver, leukæmia, pernicious anæmia, diabetes, and persistent vomiting. It is claimed that the blood may exhibit an acid reaction in cholera.

The most practical method for determining the reaction of the blood is that of Liebreich. A small plate of plaster-of-Paris, which has been previously soaked in litmus solution (neutral), is placed in contact with a few drops of the blood, which is then washed off. The reaction may then be noted.

3. **Estimation of the Percentage of Hæmoglobin.**—For this purpose one of two instruments may be used. The one which has long borne the professional confidence is the hæmometer of Fleischl, although the Dare hæmometer is rapidly becoming the favorite. The only objections to the Fleischl instrument are that it is hardly reliable when dealing with very low hæmoglobin percentages, and it is rather expensive for general use among practitioners at large.

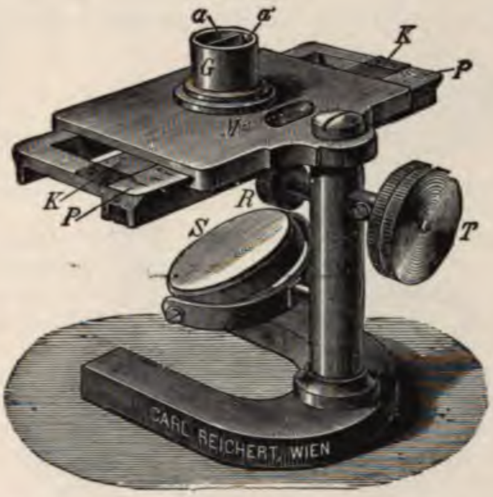
The Fleischl hæmometer (Fig. 148) consists of the following parts: A stand on which is carried a movable stage, consisting of a steel frame, in which is mounted a long glass wedge, stained with cassian purple. Opposite to the wedge the side of the frame is graduated from 0 to 120 per cent., thus indicating the quantity of hæmoglobin corresponding to the shade of color opposite to the figure. The wedge, with its carriage, is moved by means of a ratchet-screw.

Immediately over the line travelled by the wedge is placed a small reservoir having two compartments, so arranged that one is immediately over the wedge, while the other is not. The former is filled with plain water and the latter with the blood-mixture, to be described presently. Beneath the stand is a reflector of plaster-of-Paris.

In addition to the above, which constitutes the hæmometer proper, we have a number of capillary pipettes, and one of moderately large size.

To use the instrument, proceed as follows: Apply the tip of one of the capillary pipettes to the drop of blood, and the latter will enter at once by capillary attraction. Wipe off all blood clinging to the exterior of the instrument. Then fill the larger pipette with water, and with it wash the blood out of the capillary tube into the proper compartment of the reservoir. Fill the latter to its edge with the mixture of blood and water, observing due care that the top of the fluid is neither convex nor concave. Next fill the remaining compartment with plain water. We are now ready for making our observation. For this purpose, lamp-, candle- or gas-light is necessary. The Welsbach, electric, or sunlight will not do. The stage, with the wedge, is moved along until the shade of the latter, as viewed through the compartment containing plain water, corresponds with that of the mixture of blood and water. The result is noted on the graduated edge of the carriage.

FIG. 148.



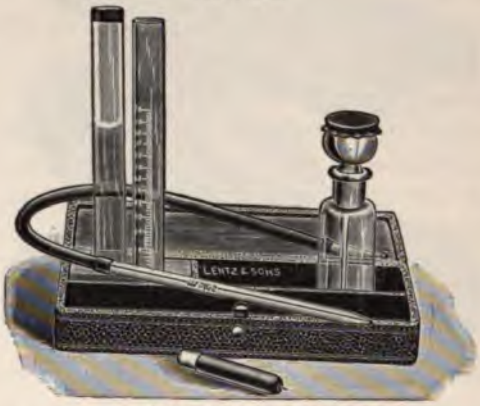
Von Fleischl's hæmometer. *a*, Compartment for blood-mixture; *a′*, Compartment for plain water; *K*, Prism of glass stained with cassian purple; *P*, frame for carrying same; *M*, Index for reading volume of hæmoglobin.

The hæmometer of Gowers is much used in England. It is hardly as accurate as Fleischl's instrument; but it is inexpensive, and hence is within the means of most general practitioners. This instrument consists of the following parts:

On a stand are mounted two upright tubes. One of these contains a solution of picrocarmine glycerin, and is hermetically sealed. The other is graduated in 134 divisions, each of which represents 20 cubic millimetres. The picrocarmine solution corresponds in color to a 1 per cent. mixture of normal blood in water. For the purpose of measuring a definite quantity of blood, there is a pipette marked at 20 cubic millimetres. To use the instrument, proceed as follows:

With the pipette take from the exuding blood just 20 cubic millimetres. After wiping off any blood that may be clinging to the exterior, blow the contents into the graduated tube, taking care that none of the blood is deposited on the walls. To make sure that all the blood is used, draw some water into the pipette and mix this with the blood in the graduate. Now hold a piece of white paper behind the upright tubes, and pour water into the graduate until the color of the mixture corresponds with that of the picrocarmine glycerin. The quantity of mixture in the tube represents the percentage of hæmoglobin as compared with the normal standard.

FIG. 149.



Gowers' hæmometer.

The **Dare Hæmoglobinometer,** although a new instrument, has already won its way as the most convenient as well as the most satisfactory in its results of any instrument in its class. Its special advantages lie in the use of undiluted blood and the speed with which the test may be made without sacrificing accuracy. The essential parts of the apparatus are an automatic pipette for collecting the blood and a graduated color comparison to measure the percentage of hæmoglobin.

Fig. 150 is composed of an oblong plate of white glass (A) of standard color, into the end of which is ground a depressed surface of measured depth, the floor being exactly parallel with the plane surface of the glass. This depression forms a shallow capillary chamber (D) when the transparent glass (B), that completes the pipette, is firmly clamped upon it by a pipette clamp (C), and which fills automatically by capillary attraction when either of the three free edges is touched to the blood drop.

The Pipette Clamp is a metal frame with two parallel bars, between which the pipette glasses are firmly held together by a bone-tipped thumbscrew working in the upper bar, the point of which makes pressure upon the centre of the white glass, preventing tilting of the edges from uneven compression. The pipette is held in position for examination by guides on the stage of the instrument, which run in grooves on the lower bar of the clamp (C′).

FIG. 150.

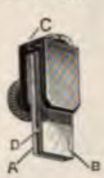


FIG. 151.

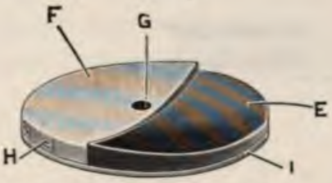


Fig. 150.—The Automatic Pipette for Collecting the Blood. *A.* White glass of pipette. *B.* Transparent glass of pipette. *C.* Pipette clamp, showing milled screw which makes pressure over centre of white glass, preventing tilting from uneven pressure. *C.* Groove into which corresponding guides on stage of instrument slide, holding pipette firmly in position. *D.* Capillary chamber of measured thickness into which the blood is drawn automatically by capillary attraction.

Fig. 151.—Color Comparison Scale. *E.* Prism of colored glass. *F.* Semicircle of white glass, the edge carrying the index of hæmoglobin percentage etched and filled-in with black; this edge also serves as a friction-surface for the rubber-covered roller by which the prism is rotated. *G.* Hole in which hub is fixed with rubber washers, which lessen liability of breaking. Pivots in centre of case hold the prism firmly in place, allowing free rotary but no lateral motion. *H.* Index of hæmoglobin percentage etched in black, showing very distinctly. *I.* Disk of white glass, which serves the same purpose as in the blood pipette, breaks the glare of direct light, and furnishes a white background to view the shades of color.

The Graduated Color Comparison. Fig. 151 is a fixed color scale representing the hæmoglobin percentage from 10 to 120 per cent.

The tinted and white glass semicircles are secured to a thin white glass disk (I) of standard color, which serves an identical purpose as in the blood pipette, diffuses the direct rays of light, and insures a constant white background, against which color shades are best appreciated.

The camera tube (U, Fig. 153) is a telescoping cylindrical tube, the internal surfaces being grooved to prevent reflection. The outer end is closed with convex lens, which magnifies threefold the color apertures that cover a surface about equal to 3 per cent. of the comparison disk.

The candle and the direction of its flame are held vertically; the color fields are then on a horizontal plane, and are viewed by the internal and external quadrants of the eye, which are equally sensitive, inasmuch as there is a frequent variance of the sensibility of the upper and lower halves of the retina.

As the comparison should quickly follow the filling of the pipette, the instrument is prepared for immediate observation by locking the camera tube, by a turn, into the threaded socket of the movable wing, and placing the candleholder in the mortise from the opposite side. The candle should then be pressed into the spring at such a level that the blue flame of the candle is below the color apertures, care being taken to have the wick of proper length (half-inch), not too short to give the desired degree of illumination, and not charred at the tip. Curved or eccentric wicks should be turned so that the intensity of light in a vertical position is midway between the two comparison apertures, which are held on a horizontal plane.

FIG. 152.

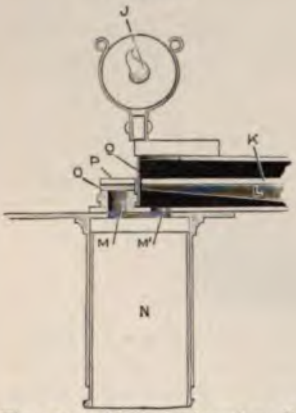


FIG. 153.

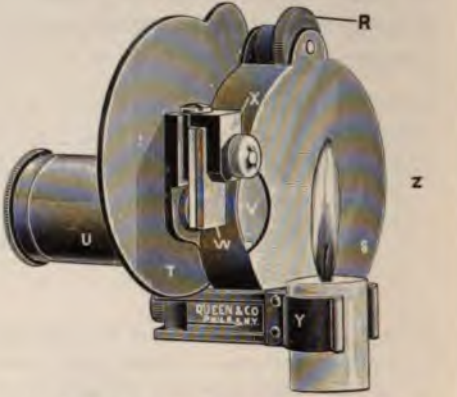


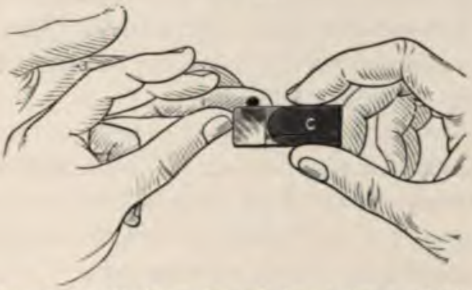
Fig. 152.—Horizontal section of instrument on a level with centre of comparison apertures. *J.* Candle; curves in wick should be pointed towards instrument and junction of the springs of candleholder to insure equal illumination of blood-film and color prism. *K.* White glass disk of color prism. *L.* Color prism. *M.* Aperture through which color of blood-film is viewed. *M'.* Aperture through which the illuminated color prism is viewed. *N.* Detachable telescoping camera tube through which the colors, placed horizontally, side by side, are viewed. *O.* Transparent glass of pipette. *P.* White glass of pipette showing section of blood-film between *O* and *P* *Q.* Metal septum between blood-film and color prism.

Fig. 153.—Instrument ready for use; illustration one-half actual size. *R.* Milled wheel by which the color prism is rotated by friction exerted upon its edge. *S.* Case enclosing color prism, showing stage upon which the blood pipette slides. *T.* Movable wing pivoted to case. When drawn outward, screens the eyes of observer from the light. When not in use, lies superimposed upon the circular prism case, occupying no extra space. *U.* Telescoping camera tube in position for examination. *V.* Opening in prism case, admitting light for illumination of color prism. The white glass disk of prism is seen inside. *W.* White glass of blood pipette. *X.* Pipette clamp held in position on the stage by grooves and guides. *Y.* Detachable candleholder. *Z.* Rectangular opening in edge of case for reading hæmoglobin percentage indicated by bevelled blade.

The pipette is then removed from the stage. The glass plates are released from the clamp by turning backward the thumb-screw, and polished with a dry towel or tissue-paper. If recently exposed to low temperature this is a matter of importance, as condensation may cause a moisture to form which retards the automatic filling of the pipette; moreover, the glasses should be moderately warm, and the inside surfaces not touched after polishing, as a greasy surface favors breaking up of the blood-film. When the plates have been washed in alcohol and polished, retraction of the edges is greatly retarded. After cleansing and polishing the white glass it is laid aside, with the blood-cavity uppermost, until the transparent plate has been prepared in a like manner and placed upon it. The two glasses are then grasped by the edges and inserted into the clamp (the screw tip against the white plate); using the fingers as a guide, the screw is set with considerable but not undue pressure, and the pipette is ready for use.

The part which has been cleansed and rendered slightly hyperæmic by the operation is steadied by the thumb and finger of the left hand and punctured; the first drop of blood is wiped away with a dry cloth, while the second, which should correspond in size to a drop of water, is touched, as it emerges from the wound, to either of the three free edges of the pipette, held horizontally, which fills automatically to its margin by capillary attraction; when filled, the excess upon the edge presented to the blood-drop is removed by quickly wiping it as the pipette is withdrawn. The surfaces of the plates should be inspected, and smears at once removed; blood adhering to the edge may remain, as it is out of the field of observation. The pipette is then placed in the instrument and the reading taken.

Fig. 154.



Manner of filling the pipette. *C.* Under surface of pipette clamp, which is held on a horizontal plane; the filling of the pipette to its margin is observed through the transparent plate. Capillary attraction and not gravitation fills the pipette.

The camera tube, always horizontal, is drawn out until focussed to give clearly-defined cold fields; the candle and the direction of its flame should be kept vertical. Draughts must be avoided, as the wavering flame sheds unsteady light, and precludes the possibility of accurate comparison. If the blood in the pipette should become coagulated, the camera tube must be thrown out of focus and the observation made in this position.

The comparison should be made by short impressions, not exceeding ten seconds, rather than by observations of long duration. When the blood and color fields are in accord, the eyes should be rested for a few seconds on the green lining of the leather bag in which the instrument is packed, and a final judgment of comparison made.

When the examination is conducted in a darkened room the readings are more uniform.

When the examination is made in daylight the candle is held close to a dark, lustreless surface, as a black coat, that does not reflect light, the line of vision directed in such a way that direct or reflected light is not permitted to enter beside the candle flame. If, however, the blood and color fields are not of the same color, the violet rays of the solar spectrum are not wholly occluded by the yellow light of the candle flame, the position should be changed until the fields appear of the same color. It is in daylight observations that the movable screen finds its most important use. The eye once applied to the eyepiece of the camera tube may not be removed until the colors are perfectly matched.

In reading the index with a view to comparing successive trials, the right hand held a few inches from the index in the edge of the case of the instrument as it is rotated, so that it comes into view, screens the eyes from the light and reflects it upon the index.

The reading is indicated by the bevelled edge of the rectangular opening (Z, Fig. 153), the percentage beneath this edge representing the amount of hæmoglobin contained in the blood examined, which is represented in percentages of the normal. If the reading is 100, the hæmoglobin is normal; if, however, the reading is 70, there is 70 per cent. of the normal, or 30 per cent. less than normal; if the reading is 120, there is 20 per cent. more than normal. While the normal percentage for male adults is 100 per cent., that of healthy females will be somewhat less,—85 or 90 per cent.

Noting the result of the readings concludes the examination. The pipette is then removed, cleansed, and returned to the stage, as already suggested. The blood is removed with greater ease when not allowed to dry upon the pipette plates.

A ready and cheap apparatus for the determination of the hæmoglobin percentage, and which is popular with some authorities, is the **Tallquist hæmoglobin scale.** This consists of a series of plates colored to correspond with that of various blood-specimens containing from 10 to 100 per cent. of hæmoglobin. The scale is accompanied by a book of white filter-paper. A piece of the latter is dipped into the blood, and as soon as the gloss has disappeared, but before drying, it is compared with the color-scale, and the one to which it corresponds most closely in color, represents the percentage of hæmoglobin.

Significance of Alterations in the Percentage of Hæmoglobin. —A diminished percentage of hæmoglobin is known as *oligo-chromæmia.*

Naturally, the hæmoglobin percentage is diminished in all diseases in which there is a lessening of the number of red blood-cells. Nevertheless, the relation between the blood and count and the percentage of coloring-matter is not a fixed one, for there are certain diseases, notably chlorosis, in which the loss of hæmoglobin is relatively greater than the diminution in the number of red blood-cells; and, on the other hand, there are others, of which pernicious anæmia is an example, in which the hæmoglobin percentage is greater than the number of red cells present would lead us to expect.

The relation between the hæmoglobin percentage and the number of red blood-cells is known as the **color-index of the blood.** Normally, the color-index is stated as 1. The number of red cells in a cubic millimetre is normally 5,000,000. If, in a given specimen, the blood-count is found to be 4,000,000, while the hæmoglobin percentage is 60, the color-index is 0.75. The method by which this result is obtained is as follows: 4,000,000 red cells per cubic millimetre represent 80 per cent. of the normal count. We make this figure the denominator of our fraction. The numerator is the hæmoglobin percentage. Our fraction, therefore, is $\frac{60}{80}$, which, reduced to decimals, equals 0.75. Take a case of pernicious anæmia as another example, for this is practically the only condition in which we find the color-index increased. The blood-count, say, is 1,000,000; the hæmoglobin, 30 per cent. We have as our fraction $\frac{30}{20}$, which, reduced to decimals, gives 1.5 as the color-index.

As already stated, the color-index of chlorosis is always low. It is also low in the anæmia of malignant disease, but not as markedly so as in chlorosis. Hæmoglobin investigations may be utilized in determining a conclusion in cases in which malignant disease is suspected. For example, an obscure gastric affection, with gradually diminishing hæmoglobin percentage, in a person past the age of forty, providing the associated symptoms do not contra-indicate such a suspicion, may well give rise to thoughts of cancer of the stomach. It is an interesting point, though not of much diagnostic value, that the hæmoglobin regeneration after operations for malignant disease is slower than after operation for the removal of non-malignant tumors.

Hæmoglobin is reduced in typhoid fever, and during convalescence may fall to a remarkably low figure.

In diseases characterized by increased watery elimination, as diabetes and acute diarrhœa, both hæmoglobin percentage and blood-count are increased, because of the lowering of the proportion of the watery elements of the blood by the dehydration. In such cases, however, the increase in both particulars runs parallel, and the color-index remains normal.

In pernicious anæmia, on the other hand, there is great diminution of the number of red blood-cells, many of which are of abnormally large size, and hence must contain a larger amount of hæmoglobin than they would were they normal. While the diminished number of blood-cells brings about a diminished hæmoglobin percentage, the increase in their size the greater amount per cell, makes that reduction less than it would otherwise be; hence the color-index is higher than normal.

Hæmoglobinæmia.—This is a condition in which the hæmoglobin leaves the red blood-cells and is dissolved in the blood-serum. It is associated with choluria, and sometimes with hæmoglobinuria. Its presence may be determined by the following procedure: A small quantity of blood is withdrawn by cupping-glasses and placed in a vessel on ice for twenty-four hours. By this time there will have appeared a shrunken clot floating on serum. In the case of normal blood the serum will be cleared, while in hæmoglobinæmia it will be of a ruby-red color. Heating this serum to a temperature of 80° C. will produce a more or less brown coagulum.

Hæmoglobinæmia may be produced by certain poisons, notably chlorate of potash, carbolic acid, arsenic, sulphide of antimony, antifebrin, antipyrin, phenacetin, sulphonal, and tincture of iodine when given hypodermically. It occasionally occurs during the course of acute and chronic infectious diseases, notably in scarlatina, intermittent fever, syphilis, variola, and scurvy. It has also been observed in Raynaud's disease, and after the transfusion of blood from one mammal into that of another.

4. **The Determination of the Time of Coagulation.**—This method part of the examination has but little importance from a diagnostic standpoint. Its chief utility resides in the treatment of cases of hæmophilia, when, by making comparisons from time to time, one is enabled to judge of the results of his treatment. Two methods for determining the coagulability of the blood have been suggested: 1. The glass-slide method; and, 2. Wright's tubes or coagulometer.

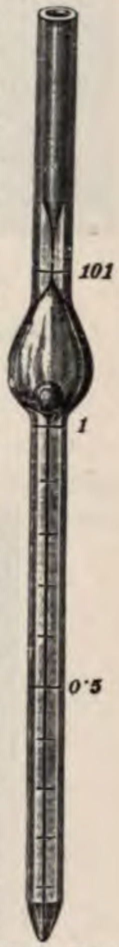
1. **The Glass-Slide Method.**—Six or seven distinct and separate drops of blood are placed upon a glass slide. A broom-straw is then taken, and at regular intervals of one minute is drawn through one of these drops. When, finally, it is noted that a thread of fibrin is clinging to the straw, the reaction is completed and the time noted. With normal blood, this should be in from two-and-a-half to five minutes.

2. **Wright's tubes** or **coagulometer** consists of a central tin receptacle for warm water (temperature 99° F.), surrounded by nine pockets, in which are placed a thermometer and eight coagulation tubes. The latter are 10 cm. in length and 0.25 mm. in diameter, and are open at both ends. To apply the method, the central tin is filled with warm water at blood-heat, and each of the tubes is filled with the blood from the patient's finger by suction. After the first tube has been filled for three minutes, an attempt is made to blow out its contents upon a sheet of paper. The other

tubes are tested in the same way at intervals of one minute, until one is reached from which the blood cannot be blown out. This is to be regarded as representing the coagulation time. Normally, this should be from three to five minutes.

5. **Determination of the Number of Red Blood-Cells Per Cubic Millimetre of Blood.**—The most convenient apparatus for this purpose is the Thoma-Zeiss hæmatocytometer. This instrument (Figs. 155 to 157, inclusive) consists of the following parts:

FIG. 155.



Mixing Tube.

(*a*) A **capillary pipette** or mixing-tube (Fig. 155) with a bulbous enlargement in which lies, free and movable, a small glass ball. The long stem of this pipette is pointed, and is marked with ten subdivisions, the figures 0.5 and 1.0 appearing at the fifth and tenth subdivisions respectively. Above the bulb appear the figures 101. To the distal extremity of the pipette is attached a small, soft-rubber tube with mouthpiece, which is used for drawing the blood-specimens into the instrument.

(*b*) A **counting-chamber**, which is exactly 0.1 millimetre in depth. The floor of this chamber or cell is divided off by ruled lines into spaces so that the quantity of material overlying each equals exactly $\frac{1}{4000}$ of a cubic millimetre. Heavy lines are ruled at such intervals as to include within them sixteen squares.

To use the instrument, the finger or lobe of the ear is punctured, and a portion of the exuding blood is drawn into the pipette to figure 0.5 or 1.0. Any blood clinging to the point is carefully wiped off, and then the instrument is filled to the mark 101 with a solution of common salt (3 per cent.). We now have in the bulb a mixture of blood and salt solution in the proportion of 1 to 200 or 1 to 100, according as the stem is filled with blood to 0.5 or 1.0 respectively. The pipette is next shaken carefully, the free ball in the bulb aiding in the admixture of blood and diluent.

Other fluids than 3 per cent. salt solution may be used as a diluent. **Toison's fluid** has always been deservedly popular, because it stains the leucocytes and renders them easily visible. Its composition is as follows:

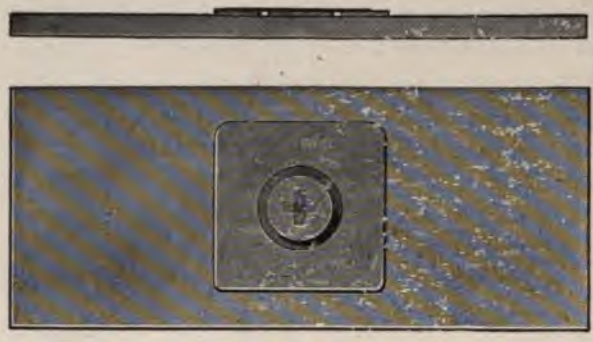
Distilled water,	160	parts.
Glycerin,	30	parts.
Sodium sulphate,	8	parts.
Sodium chloride,	1	parts.
Methyl violet,	0.025	part.

Still other fluids recommended include Hayem's and Pacini's fluids, the formulæ of which are herewith appended.

Pacini's fluid:

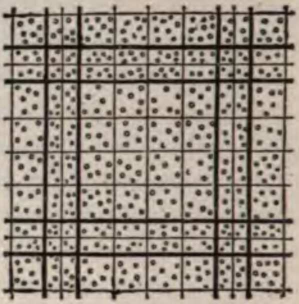
Bichloride of mercury,	2.0 grammes.
Sodium chloride,	4.0 grammes.
Glycerin,	26.0 grammes.
Distilled water,	226.0 grammes.

FIG. 156.



Microscope slide with counting-chamber.

FIG. 157.

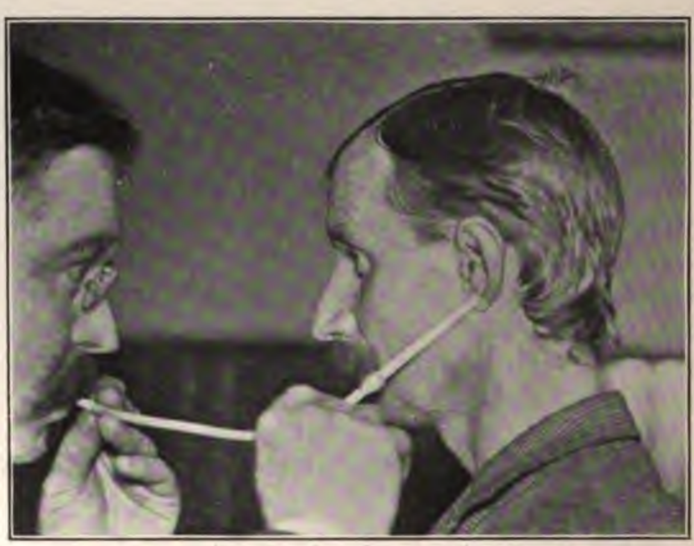


Microscope field showing red corpuscles in squares.

Hayem's fluid:

Bichloride of mercury,	0.5 gramme.
Sodium sulphate,	5.0 grammes.
Sodium chloride,	2.0 grammes.
Distilled water,	200.0 grammes.

FIG. 158.

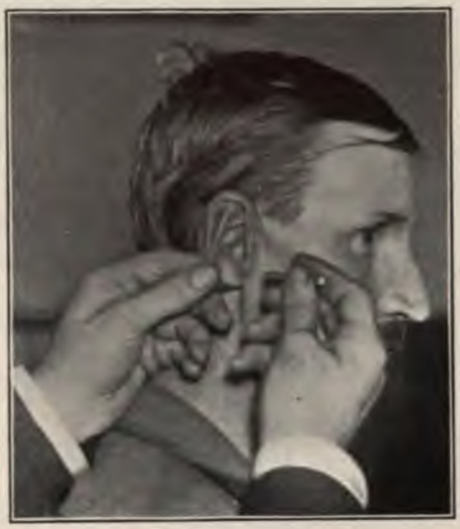


The wrong position and the one in which the majority of students hold the pipette when withdrawing blood.

Another solution of use for diluting the blood when counting the red blood-corpuscles is **Gowers' fluid.** Its particular advantage lies in the fact that it destroys the leucocytes. Its formula is as follows:

Sodium sulphate,	104 grains.
Acetic acid,	1 drachm.
Distilled water, ad,	4 ounces.

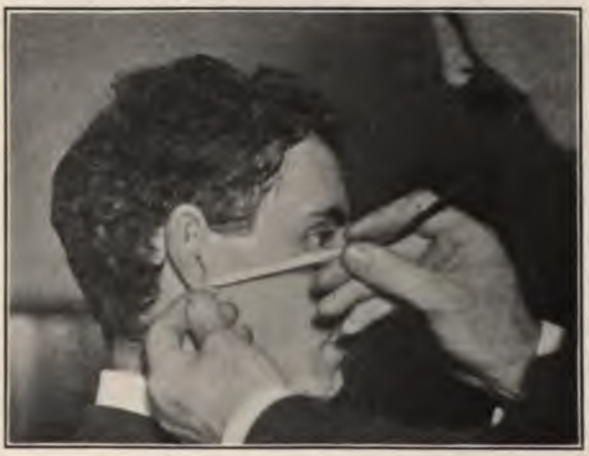
FIG. 159.



Position for holding ear and lancet when obtaining a blood-specimen.

The blood having been thoroughly diluted with one of the above-named solutions, the fluid remaining in the stem of the pipette is discharged, as that contains no blood. Then a drop of the mixture is carefully deposited in the counting-chamber. The cover-glass is next adjusted, care being taken that no bubbles of air shall enter. If this is done properly, Newton's

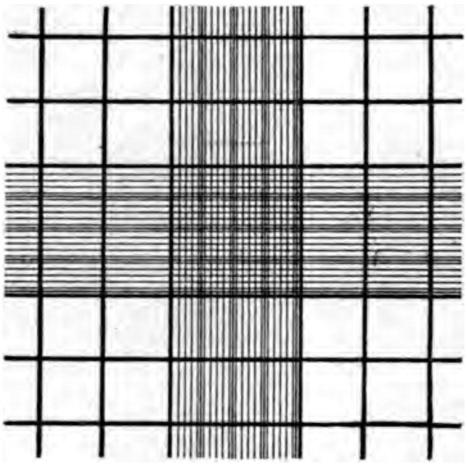
FIG. 160.



The proper position for holding the pipette when drawing blood into the instrument.

color-rings will be visible at the margins of the drop. The instrument is then set aside for a few minutes to permit the corpuscles to settle, when it is placed under the microscope, an objective of low power being used, and the number of corpuscles in from 80 to 160 squares counted. From the result thus obtained the number of red corpuscles in a cubic millimetre of blood may be calculated. It is important to bear in mind that liability to error decreases as a larger number of squares is counted. To avoid counting the same corpuscle twice, it is well to make it a rule of procedure that all corpuscles touching or overlying the right hand and upper boundaries of a square shall be included in the count of that square. The calculation of the number of corpuscles per cubic millimetre is based upon the fact that each square has overlying it $\frac{1}{4000}$ of a cubic millimetre, and the blood-mixture is in the proportion of 1 to 100 or 1 to 200, as the case may be. Our calculation is then as follows :

FIG. 161.



Zappert's modification of the Thoma-Zeiss counting-chamber.

Multiply the number of corpuscles counted by 4000, and this result in turn, by 100 or 200, as the dilution was 1 to 100 or 1 to 200. Divide the result by the number of squares, and we have the number of red cells per cubic millimetre. For example, if we find in 80 squares 200 corpuscles, and the degree of dilution was 1 to 200, we multiply the 200 by 4000, which gives us 800,000; this, in turn, we multiply by 200, and we get 160,000,000. Dividing by 80, the number of squares counted, and we have 2,000,000 red corpuscles per cubic millimetre of blood.

Zappert has modified the Thoma-Zeiss counting-chamber by adding extra lines so as to increase the ruled area to nine times its original size. Thus divided, the chamber presents 9 large squares, each equal in size to the central group of 400 small squares, and, as a result, the entire ruled surface equals the area of 3600 of the latter. To further simplify the process, the peripheral squares are subdivided into 4, each of which has the same area as 100 of the small squares. This counting-chamber greatly simplifies the counting of leucocytes, and is coming into use in preference to the older instrument.

6. **To Count the Number of Leucocytes per Cubic Millimetre of Blood.**—For this purpose we also use the Thoma-Zeiss apparatus, but with a special leucocyte pipette, in which we dilute the blood in the proportion of 1 to 10 or 1 to 20, as we prefer. The pipette has a much larger bore than the one used for red corpuscles. The quantity of blood required is correspondingly larger. It should be diluted with a one-third of 1 per cent. solution of glacial acetic acid, which destroys the red cells, while not affecting the white. Hence the latter alone are visible. The count is made on exactly the same principle as that of the red cells. Owing, however, to the small number present, a very large number of squares should be counted. It should also be borne in mind that the degree of dilution is 1 to 10 or 20, not 1 to 100 or 200.

Clinical Significance of Alterations in the Corpuscular Counts.—A diminution in the number of the red blood-corpuscles has no diagnostic significance, *per se*, inasmuch as all forms of anæmia are characterized by this symptom. Then, too, any general depreciation of the bodily health is associated with a lessening of the corpuscular richness of the blood. Various diseases, however, have markedly different effects in this particular. Thus, progressive pernicious anæmia is attended by a most remarkable diminution in the number of red blood-cells (oligocythæmia), no other disease being comparable to it in this respect. Blood-counts of less than 2,000,000 are not uncommon in the early stages, and one extreme instance in which it was but 360,000 has been recorded. In chlorosis the count is remarkably high as compared with the usual pallor of the patients, depreciations of corpuscular richness not averaging more than 40 per cent., the majority of cases showing not less than 3,750,000 red cells per cubic millimetre.

While the blood-count, then, possesses but little diagnostic value, it is invaluable as a means of judging of the general condition of the patient, the actual pathological condition having been determined by other means. Repetition of blood-counts from time to time is likewise important as affording us positive knowledge of the progress of the same.

An estimation of the number of leucocytes possesses considerable diagnostic value, *per se*, as there are certain diseases which are characterized by a hyperleucocytosis of great or small degree. Concerning this subject, more will be said after studying the microscopic features of the leucocytes in health and disease.

7. **Examination of Dried and Stained Blood-Films.**—This constitutes by all odds the most valuable of procedures in determining the condition of the blood. It is one, moreover, requiring experience and attention to minute details. At the same time the technique is simple, and can readily be grasped by any practitioner who is determined to give the time necessary for carrying it out. The most important part of the process, the one concerning which the most mistakes are made, is the first, namely, that of preparing the blood-film. For this a *sine qua non* is clean cover-glasses. Without such, a properly prepared film is an impossibility.

The cover-glasses employed should be of very thin glass ; square ones are better than round, because more conveniently handled. As they come from the shop, they are covered with a closely adherent dirt. To remove this, the slips should be washed first in ether, and then in absolute alcohol, and wiped carefully with a soft silk handkerchief. After wiping, they should never be handled excepting with properly-constructed forceps. Under no circumstances should they be manipulated with the fingers. Cabot recommends that they be washed in water after having been smeared very liberally with soap. This certainly takes off all the macroscopic dirt, but is not as satisfactory as ether and alcohol. After cleaning and drying, the cover-slips should be placed in a dust-proof receptacle ready for use.

To prepare the blood-film, puncture the finger or lobe of the ear. Wipe off the first few drops of exuding blood. Then apply a cover-slip very carefully to the apex of the drop of blood. Under no circumstances must the glass be permitted to come in contact with the skin. The mistake usually made is that of getting too large a quantity of blood on the slip. The specimen thus obtained is next brought in contact with another cover-glass, as in Fig. 163, and the blood permitted to spread itself in a thin layer by the weight of the glass alone. It is important that no pressure be exerted to hasten the spreading process, as such a procedure must cause distortions of the corpuscular elements of the blood. If the glasses are properly cleansed, the spreading will take place evenly and in a thin layer. Thinness of the layer is important, because, if the blood is not properly spread, corpuscles will be superimposed upon each other to an extent making it impossible to determine the structure of more than a few cells, and will interfere seriously with the staining and fixing process. If the spread between the cover-glasses should reach the edges of the latter, it is a settled fact that that particular spread will be useless. It makes no difference how small a quantity of blood is obtained, for the essential factor is not to get too much.

The next step is that of separating the cover-slips. This should be done by sliding them apart horizontally. To secure a thin and even layer, this separation of the cover-slips must be done with great rapidity. If the movements are slow, the cells will be massed together and the spread

Fig. 162

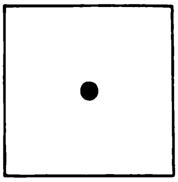


Fig. 163

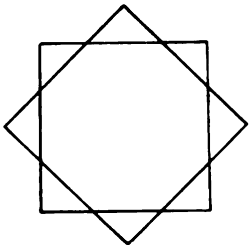


Fig. 164

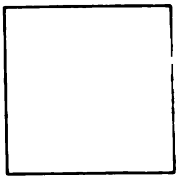
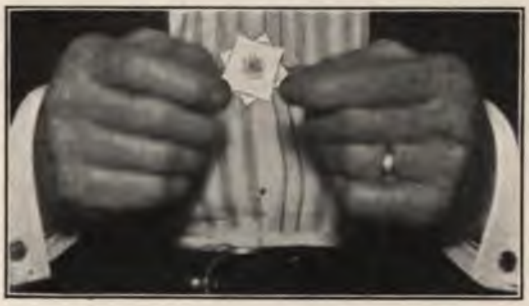


Fig. 162. Drop of blood as it appears when first placed on cover-slip. Fig. 163. Appearance of properly spread film. Fig. 164. Blood-smear after separation of the cover glasses.

uneven. They should then be permitted to dry in the air, when they may be laid aside, protected from dust, until the physician has the leisure to examine them systematically. Blood-films thus prepared will keep a long time. It has been recommended that they be preserved between layers of thin blotting-paper. It would seem, however, that placing the films face to face, and wrapping in fresh tissue, would be a better method, it being presumed that the film surfaces of specimens of the same case only will be brought in contact.

Some prefer to make their spreads on slides instead of using cover-slips. A small drop of blood is deposited upon one end of the slide. Another slide is then brought in contact with it at an acute angle. This position is to be maintained until the drop of blood has spread itself along the entire line of contact of the two slides. Then, with one quick sweep, the spread is thinly and evenly made. After drying in the open air, the slide may be examined under low power, and the portion found best adapted for staining by reason of the thinness of the spread selected for staining.

FIG. 165.



Position for holding cover-glasses when separating them to make the blood-smear.

Fixing the Specimen.—The best method of fixation of the blood-films continues to be that first proposed by Ehrlich, namely, the heating of the same at a temperature of 110° C. for from one-half to one hour. The method suggests the use of a special apparatus in order to maintain the standard heat. As a matter of fact, such is by no means necessary. It has long been known that when a copper-plate has been exposed to a Bunsen burner or alcohol flame for ten minutes or thereabouts, the temperature at definite distances from the central heating-point remains constant for that particular point, heat formation and heat dissipation being equal. By dropping water along a line towards the centre, one finally reaches a spot at which the water boils. Just inside of this spot the temperature approximates 110° C. for all practical purposes, and here we may place our cover-slip preparations. It is claimed that for ordinary examination

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a fifteen-minute fixation is all-sufficient. If one wishes the best result, he should continue the process to the maximum, *i.e.*, one hour. He should also bear in mind that the longer the specimen is heated, the longer will be the time required for staining.

A very convenient and quick method of fixation is that of placing the cover-slip in forceps, and then passing it through an **alcohol or Bunsen flame** eight or ten times. This method, if carelessly practised, may destroy the specimen ; hence it is not to be advocated unless the operator has had some experience.

Da Costa has devised an oven for the hardening of blood-films by heat. It consists of an oblong, shallow copper box with a thick bottom, mounted on an iron stand, and provided with a thermometer registering to 200° C. Its low price puts it within the reach of all who do clinical laboratory work.

Next to Ehrlich's method is **fixation by alcohol and ether.** The film is placed in a mixture of equal parts of absolute alcohol and ether for one-half hour, or as much longer as may suit the convenience of the physician.

For very rapid fixation, a one per cent. **alcoholic solution of formol** is excellent. Place a few drops of this on the blood-film ; let it remain for a minute or so, and then pour it off.

Staining Blood Specimens.—Many formulæ have been proposed for the staining of blood specimens, each of which is supposed to have special advantages. Those most generally used are Ehrlich's tri-acid stain and eosin, and Delafield's hæmatoxylon. The formulæ of the various staining solutions are here appended, not because the physician is recommended to have them prepared to his order, but because it is well to know their composition. The staining of dried-blood specimens has now become so well-recognized as a part of a clinical examination that all the stains in common use are kept in stock by the large supply houses, and can be purchased at prices ranging from twenty-five to fifty cents for a bottle containing thirty cubic centimetres. Those prepared by Grübler, of Germany, are generally admitted to be the best.

Ehrlich's Tri-Acid Stain.—Saturated watery solutions of orange G, acid fuchsin and methyl-green are first prepared. Before mixing they must be permitted to stand not less than one week, in order to clear. The various ingredients are then mixed in the order given in the following formula :

Orange G solution,	13–14 c.c.
Acid fuchsin solution,	6–7 c.c.
Distilled water,	15 c.c.
Alcohol,	15 c.c.
Methyl-green solution,	12.5 c.c.
Alcohol,	10 c.c.
Glycerin,	10 c.c.

The formula recommended by Cabot is as follows:

Orange G solution,	6 c.c.
Acid fuchsin solution,	4 c.c.

To these add a few drops at a time, shaking between each addition:

Methyl-green solution,	6.6 c.c.

Then add:

Glycerin,	5 c.c.
Absolute alcohol,	10 c.c.
Distilled water,	15 c.c.

The writer uses the preparation made by Grübler, and on sale in the instrument shops.

Ehrlich's tri-acid solution stains the nuclei of the leucocytes greenish; the eosinophilic granules a copper color; the neutrophilic granules violet; the nuclei of the basophilic leucocytes a pale green; and the red corpuscles orange, excepting in cases of chronic anæmia, when they assume a mixed tint, in which fuchsin predominates more or less.

Eosin and Hæmatoxylin.—This is a useful contrast stain, but can hardly be considered available when one wishes to study the granulations of the leucocytes. The film is first stained in a watery solution of eosin for from thirty seconds to one minute. The eosin is then washed off, after which Delafield's hæmatoxylin is applied for a like period of time. This is then washed off, and the specimen dried between layers of filter-paper. It may then be mounted in xylol-balsam. Or, if there is no desire to preserve the specimen, it may be mounted wet.

Chenzinsky's Solution.—The formula is as follows:

Saturated watery solution of methylene blue,	20 c.c.
0.5 per cent. solution of eosin in 70 per cent. alcohol,	20 c.c.
Distilled water,	40 c.c.

This solution should be filtered before using. The film should be kept in contact with the stain from six to twenty-four hours, at a temperature of from 37 to 40° C. (in air-tight watch-crystals).

The red corpuscles and eosinophilic granules are stained a bright-red; the nuclei and basophilic granules a deep blue, and the malarial organisms a light sky-blue.

Ehrlich's Tri-Glycerin Mixture.

Eosin,	2 grammes.
Aurantia,	2 grammes.
Nigrosin,	2 grammes.
Glycerin,	30 grammes.

In order to effect a perfect solution, the various ingredients are mixed and subjected to a temperature of 40° C. in a chamber for one week.

Neusser's Stain.—This is a modification of Ehrlich's tri-acid stain, and its formula is as follows :

Acid fuchsin solution,	50 c.c.
Orange G solution,	70 c.c.
Methyl-green solution,	80 c.c.
Distilled water,	150 c.c.
Absolute alcohol,	80 c.c.
Glycerin,	20 c.c.

This is recommended for staining the basophilic perinuclear granules of Neusser. These structures, however, are claimed to be artefacts. This question seems to be unsettled at the present time.

Eosinate of methylene blue is strongly advocated by Simon, of Baltimore, as a thoroughly practical and well-differentiating blood-stain. He considers it, after over three years of constant use, as fully equal to the tricolor stain of Ehrlich. The stain is prepared as follows : "Equal parts of a 1.2 to 1.25 per cent. aqueous solution of Grübler's eosin (yellow shade) and of a 1 per cent. aqueous solution of methylene blue are mixed in an open basin, thoroughly stirred, and set aside for twenty-four hours. The resulting precipitate is collected on a filter, dried, powdered, washed with water, again collected and dried, and can then be stored for future use. Of the dye thus prepared, a 0.5 per cent. solution in pure methyl alcohol (absolute best) serves as a stain. It is ready for use almost immediately after its preparation, and is perfectly stable."

"The specimens are stained for from three to five minutes by thoroughly covering the slides with the solution. The stain is then washed off, when the preparations are well-dried in the air (not with filter-paper), and are now ready for examination. Viewed with the naked eye, the blood-films present a dirty rose-color, unless the leucocytes should be very numerous or several days have elapsed between the preparation of the smears and the staining, in which case a more pronounced bluish tint appears. On microscopic examination, the red corpuscles are usually terra-cotta colored ; but with lamp and gaslight they present a grayish appearance, and in the case of older blood they are stained a light green. All nuclei are stained blue, the neutrophilic granules a purplish red, the eosinophilic granules a bright red, and the basophilic granules of the mast cells a fine violet ;" "and the basophilic granules of the red cells are colored blue."*

Wright's Stain.—This, like Simon's eosinate of methyl blue, is a modification of the Jenner stain. To prepare the staining fluid, make a one-half of 1 per cent. solution of sodium carbonate in water in an Ehrlenmeyer flask, and add to it 1 per cent. of Grübler's methylene blue. It is very important that all of the sodium carbonate be dissolved before the addition of the methylene blue. Next steam the solution for one hour in an Arnold steam sterilizer. This process gives the finished stain polychromatic properties, by means of which the nuclei and granules of the leucocytes are brought out more sharply. When the steaming is completed, the solution is permitted to cool. When cold, it is poured into a large dish or flask. A one to one-thousand solution of Grübler's yellowish eosin added while the mixture is stirred, until the latter loses its blue color, becomes purple in color, and a scum with yellowish metallic lustre forms on the surface, while, on close inspection, a finely-granular black precipitate appears in suspension. This usually requires about five hundred cubic centimetres of the eosin solution for one hundred cubic centimetres of the alkaline methylene-blue solution.

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* "International Clinics," Twelfth series, Volume I., p. 80.

"The precipitate is collected on a filter, and without washing is allowed to dry thereon. When thoroughly dry, a saturated solution of pure methylic alcohol is made. Three-tenths of a gramme of the dry precipitate will thoroughly saturate one hundred cubic centimetres of the methyl alcohol in a few minutes.

"The saturated alcoholic solution of the precipitate is next filtered, and to the filtrate is then added 25 per cent. of methylic alcohol, *e.g.*, to eighty cubic centimetres of the saturated alcoholic solution, twenty cubic centimetres of methylic alcohol is added.

"This somewhat diluted alcoholic solution of the precipitate is the staining fluid. It is permanent and may be kept on hand ready for use."*

After the films have dried thoroughly in the air, the stain is applied in the usual way. After remaining on the cover-slip for one minute, the film may be considered as fixed, water is added to the staining fluid, drop by drop, until it becomes semi-translucent and a reddish tint becomes visible at its margins, while a metallic scum forms on its surface. As a rule, eight or ten drops are required for a seven-eighths of an inch square cover-glass. The diluted staining fluid is permitted to remain for two or three minutes, and then the preparation is washed with water. The blood film now presents a blue or purple color. Under the microscope, the red corpuscles are found to be stained blue.

To develop the differential staining of the various elements of the blood, wash the preparation in distilled water until the better spread portions of the film appear yellowish or reddish in color. Under this process the red corpuscles become greenish, then yellowish, and finally orange or pinkish in color, depending upon the depth of the original color. This differentiation or decoloration by washing generally takes about three minutes.

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* "Journal of Medical Research," January, 1902.

When the desired color is obtained, the preparation is dried between blotters and mounted.

As a result of this staining process, "the *red cells* are orange or pink in color. Polychromatophilia and punctate basophilia are well brought out. The nucleated red cells have deep blue nuclei and the cytoplasm is usually of a blue tint.

"The *lymphocytes* have dark purplish-blue nuclei and robin's-egg blue cytoplasm, in which a few dark-blue or purplish granules are sometimes present.

"The *polymorphonuclear neutrophiles* have a dark-blue or dark lilac-colored nucleus, and the granules are usually of a reddish-lilac color.

"The *eosinophiles* have dark-blue or dark lilac-colored nuclei. The granules have the color of eosin, while the cytoplasm in which they are imbedded has a blue color.

"The *large mononuclear leucocytes* appear in at least two forms. Each form has a blue or dark lilac-colored nucleus. The cytoplasm of one form is pale blue, and of the other form is blue with dark-lilac or deep purple-colored granules, which are usually not so numerous as are the granules in the polynuclear neutrophilic leucocytes.

"The *mast cells* appear as cells of about the size of polynuclear leucocytes, with purplish or dark-blue stained, irregular-shaped nuclei and cytoplasm, sometimes bluish, in which numerous coarse spherical granules of variable sizes are imbedded. These granules are of a dark-blue or of a dark-purple color, and may appear almost black.

"The myelocytes have a dark-blue or dark lilac-colored nuclei and blue cytoplasm, in which numerous dark-lilac or reddish-lilac granules are imbedded. In leukæmia, more color differences are brought out among the leucocytes than by the ordinary methods of staining."

This method of staining has now been in use in the clinical laboratory of my service for some little time, under the care of Dr. W. H. Lyle, and has given such satisfaction that it promises to supersede all other staining processes.

Pathological Changes in the Red Corpuscles.—The alterations in the red blood-cells relate to shape, size and structure. In a number of pathological conditions the red cells may be altered very greatly in shape, assuming oval or cylindrical, dumb-bell, goblet, and indeed all conceivable forms. Such a condition is known as poikilocytosis. It is especially characteristic of pernicious anæmia, and for a long time was regarded as pathognomonic of that disease. It occurs, however, in chlorosis and other forms of anæmia, though never to the remarkable degree observed in pernicious anæmia. In any event, it is to be regarded as a serious symptom. The chlorotic cases, for example, exhibit a remarkable predisposition to thrombosis. Maragliano expressed the view, with which von Jaksch agrees, that poikilocytosis is in reality a necrosis of the red corpuscles.

Changes in Size of the Red Corpuscles.—The normal red corpuscle averages from 7.2 μ to 7.8 μ in diameter. Smaller cells measuring from 6 μ to 6.5 μ and larger ones ranging from 8.5 μ to 9 μ in diameter may be observed under normal conditions. In pathological conditions remarkable variations in size may be observed. Thus small corpuscles (microcytes) measuring from 3.5 μ to 6 μ, and large ones (macrocytes) 9.5 μ to 12 μ, appear. Still larger ones, which have been termed megalocytes, may occasionally be seen, and measure from 12 μ to 16 μ in diameter.

The pathological significance of microcythæmia must be regarded as still *sub judice*. Von Jaksch declares that their discovery is of no assistance in diagnosis, and agrees with Litten, who observed that they may rapidly appear in the blood, and as rapidly disappear. Others have contended that microcytes are but the result of post-mortem changes in the blood, or that they are due to the uniform abstraction of water from the blood. Clinically, microcythæmia may be observed in pernicious anæmia, leukæmia, pseudo-leukæmia, secondary anæmias, and to a certain extent in chlorosis.

Corpuscles of large size (macrocythæmia) occur in all severe anæmias, but especially in pernicious anæmia. But they are not to be regarded as diagnostic. Their structure is, as a rule, paler than corpuscles of normal size, because the coloring-matter is spread out over a greater bulk.

Alterations in the Staining Properties of the Red Corpuscles.—Normally, the red blood-corpuscles stain only with acid dyes, notably eosin and orange G. Under pathological conditions this power is lost, in a degree, and it is also possible to stain them with the basic stains. For example, after staining a film with eosin-hæmatoxylin, some corpuscles will be found stained by eosin only, while others manifest the blue tint of the hæmatoxylin. This condition is known as polychromatophilic degeneration, and, because it is common in various forms of anæmia—"*anæmic degeneration.*"

A peculiar alteration in the staining properties of the red cells may be observed in cases of diabetes, and is known as Bremer's diabetic blood-test. In applying it clinically, one should always use some films of normal blood for control tests. The slips are heated rapidly to 135° C., care being taken that the temperature does not exceed 140° C. They are then allowed to cool sufficiently to be handled. A slip of the diabetic blood is then placed back to back with one of the normal, and stained by immersion for one to two minutes in a 1 per cent. aqueous solution of Congo-red, freshly prepared. The specimens are then washed and dried. The diabetic blood fails to take the stain, while the normal blood is of the color of the Congo-red. It has now been authoritatively determined that this test is very reliable. The substances in the blood which give rise to the reaction are as yet unknown. It has even been claimed that it exists in the preglycosuric stage of the disease.

Nucleation of the Red Corpuscles.—Three groups of nucleated red corpuscles may be recognized, the classification being based upon their size. They include (a) normoblasts; (b) megaloblasts; (c) microblasts.

(*a*) **Normoblasts.**—The normoblast is a nucleated red blood-corpuscle of normal size. Its nucleus is about one-half the diameter of the containing cell, and usually placed to one side of it. With advancing age of the cell the nucleus may be extruded, in which condition it may be seen under the microscope as a protrusion on the body of the main corpuscle, or even free.

Normoblasts are now regarded as immature red corpuscles. They are found normally in the bone-marrow; after hæmorrhage their number in this locality is greatly increased. Their appearance in the blood indicates simply that in the processes of regeneration after hæmorrhage, or in the course of an anæmia, for example, that certain cells leave the marrow and enter the general circulation before they are properly matured.

(*b*) **Megaloblasts.**—Megaloblasts are nucleated red corpuscles of large size. Usually they are 10 μ in diameter; they may attain the size of of 14 μ to 16 μ. The nucleus is large, often occupying as much as two-thirds of the diameter of the corpuscle. It does not take the stain as readily as the normoblasts; hence is much lighter in color in stained specimens. The surrounding protoplasm takes the eosin stain well. Megaloblasts are never present in normal blood. Their presence is to be regarded as evidence of serious changes, in that they are indicative of retrogression. Their persistent presence is strongly suggestive of pernicious anæmia. In view of their diagnostic importance, the search for megaloblasts in dried-blood specimens should be painstaking, and they should not be declared absent until several films have been examined. It is only exceptionally that they are sufficiently numerous to make their discovery an off-hand matter.

(*c*) **Microblasts.**—Microblasts are nucleated red corpuscles, smaller in size than normoblasts. They are much less numerous than either of the preceding varieties. Nothing is known of them clinically.

Granular Degeneration of the Red Corpuscles.—In the blood of certain diseases to be hereafter mentioned, but never under normal conditions, may be found certain granulations in the erythrocytes. They occur alike in the nucleated and non-nucleated forms. In number they vary greatly; in some corpuscles they may be but few, appearing as coarse granules dotting the substance of the shell; in others they are more numerous, and present as a diffuse stippling. In their staining qualities they

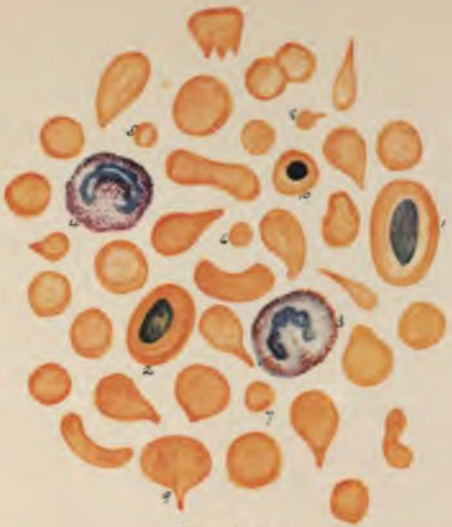

FIG. 165a.

Pernicious anæmia.

1 and 2, Megaloblasts.
3 and 4, Polymorphonuclear neutrophile
5 to 8, Microcytes.
9 Megalocytes.
10 Normoblasts.

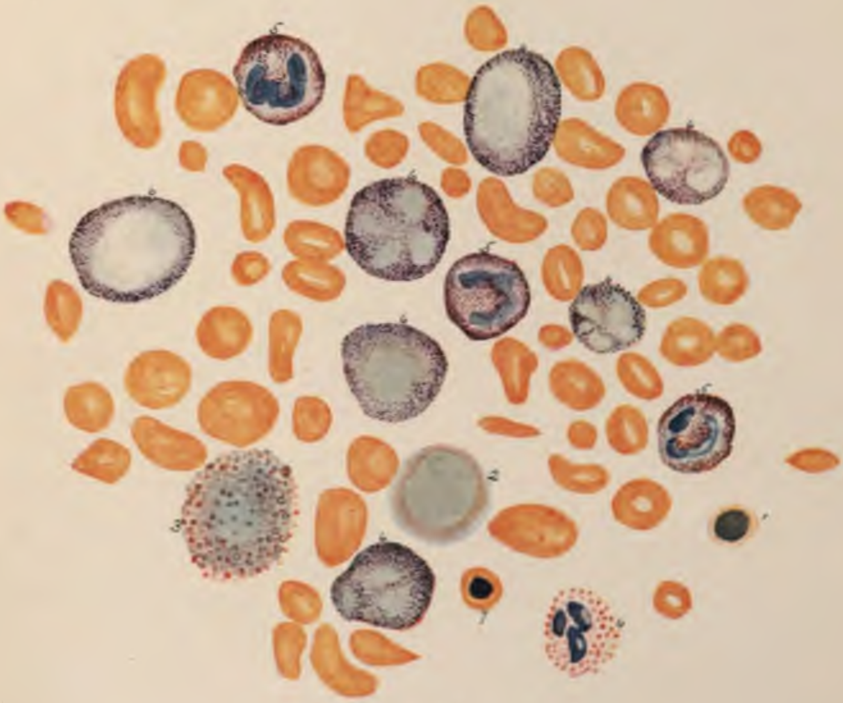

FIG. 165b.

(Triacid Stain.)

Spleno-medullary leukæmia.

1, Small lymphocyte.
2, Large lymphocyte,
3, Eosinophilic myelocyte.
4, Eosinophile.
5, Polymorphonuclear neutrophiles
6, Myelocytes.
7, Normoblast.

are basophilic. They are best brought out by Simon's eosinate of methylene blue and Wright's stain. They do not appear in films prepared with Ehrlich's tri-acid stain.

The most elaborate investigation of their clinical significance has been made by Simon. Others have studied the subject without reaching practical conclusions. Simon agrees with Grawitz, "when he states that they are found only in conditions in which hæmolytic poisons may be supposed to be operative in the body." They are found in pernicious anæmia, their numbers seeming to be in direct proportion to the severity of the case. It is even claimed that their appearance may antedate that of the other well-known blood-changes of this disease. They are rarely found in chlorosis, and then only in severe cases. They are of inconstant occurrence in leukæmia. In carcinoma they do not appear until the patient presents evidence of the cachexia, by which time it is usually a simple matter to make a diagnosis, by reason of the other symptoms present.

Simon lays especial stress upon their importance in the diagnosis of doubtful cases of lead-poisoning. His investigations have demonstrated that the condition appears within a comparatively few hours after the ingestion of small doses of lead.

The Leucocytes.—The clinical study of the leucocytes involves one of the most interesting problems in the pathology of the blood. Under no circumstances should we rest satisfied with the mere leucocyte count, but we should also determine the relative proportion of their several varieties.

The classification of leucocytes is based upon two of their structural peculiarities: (1) The character of their nuclei; and (2) the manner in which their protoplasm reacts to acid, basic and neutral stains. The following varieties are now recognized:

a. Small mononuclear leucocytes.
b. Large mononuclear leucocytes.
c. Transitional forms.
d. Polymorphonuclear leucocytes.
e. Eosinophiles.
f. Basophilic leucocytes.
g. Myelocytes.

(*a*) **Small Mononuclear Leucocytes.**—These are the smallest of the leucocytes, and they measure from 6 to 7.5 μ. They are also known as small lymphocytes, because they are supposed to have their origin in the lymph nodes. The nucleus is relatively large, and fills nearly the entire cell, leaving around it but a narrow rim of non-granular protoplasm. It stains deeply with such dies as methyl-blue and hæmatoxylin; in fact, more deeply than the nuclei of the other varieties of leucocytes. Normally, they comprise from 15 to 20 per cent. of the leucocytes present.

They are increased physiologically after eating, and decreased by starvation.

Pathologically, they are greatly increased in lymphatic leucocythæmia.

(*b*) **Large Mononuclear Leucocytes.**—The large mononuclear leucocytes measure from 10 μ to 15 μ in diameter. Their nuclei are large and of variable shape, this being accounted for by arrested amœboid movement during their preparation for examination, and to distortion by pressure. Their protoplasm is non-granular, and takes the stain poorly. In films stained with eosin and hæmatoxylin or methyl-blue, it presents a faint bluish tinge.

The large mononuclear comprise about 6 per cent. to 8 per cent. of the total number of leucocytes in healthy blood.

(*c*) **Transition Forms.**—These include leucocytes in which the nuclei are indented and distorted so as to simulate those of the class next to be considered, and at the same time the protoplasm presents some few neutrophilic granules.

(*d*) **Polymorphonuclear Leucocytes.**—They are also known as multinuclear leucocytes. They are characterized by the possession of a nucleus of most irregular shape. Owing to the twists and turns taken by the nucleus, it may give the appearance of being multiple when viewed from certain standpoints. They vary in size from 9.5 μ to 7.5 μ in diameter. Their granules are neutrophilic, and surround the nucleus completely. With the triple stain they present a lilac or blue color. With the double staining by eosin and hæmatoxylin the protoplasm stains pink, though the granulations are not evident. The substance of the nuclei is not homogeneous, and hence it does not stain regularly.

The polymorphonuclear leucocytes constitute 64 per cent. of the leucocytes in normal blood. Some authorities have stated the percentage to be as high as 70 per cent. to 75 per cent. They are generally regarded as the mature or fully-developed leucocytes.

(*c*) **Eosinophiles.**—The main distinction between the eosinophiles and the preceding variety of leucocyte is found in the staining qualities of their granules, which assume a bright pink or red color to acid dyes, as eosin or acid fuchsin. Their nuclei are polymorphous, and stain more evenly than, though not as deeply as, the polymorphonuclear leucocytes. In size they range from 9.5 μ to 11 μ. They are found in healthy blood in small numbers only,—about 2 per cent. of the leucocytes. By many they are regarded as "over-ripe" polymorphonuclear neutrophiles. They are generally abundant in areolar tissue and bone-marrow.

Considerable clinical interest is attached to alterations in the eosinophile percentage, because of the significance attached to their variations. In some diseases, as leukæmia, their absolute number is increased, while their relative proportion to other leucocytes is maintained. They are in-

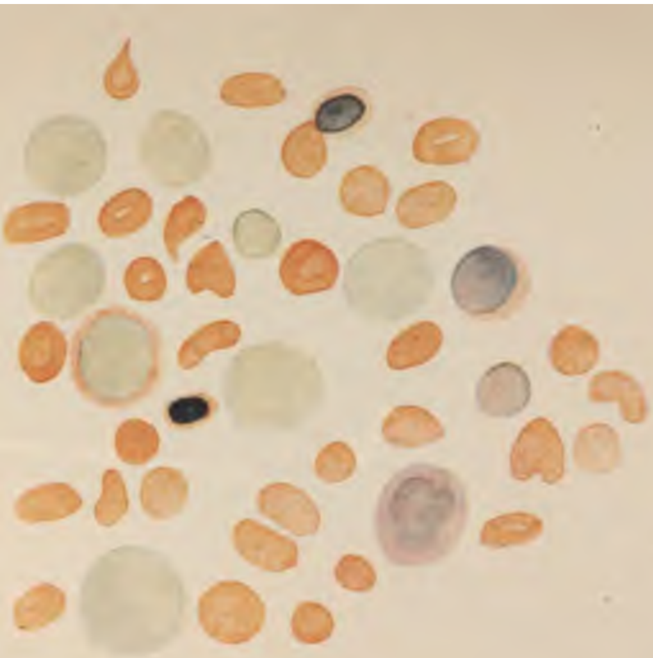

Fig. 165d.

The legend lines explanatory of figures 165*c* and 165*d*, through a mistake of the printer, were transposed. The reader will please note this correction.

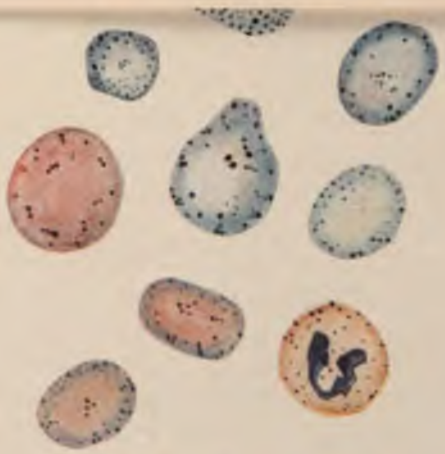

Fig. 165c.
Lymphatic leukæmia.

creased absolutely and relatively in chlorosis, bronchial asthma, infancy, trichinosis, some bone diseases, and skin diseases. Ordinarily, however, they do not increase to more than 10 per cent. to 20 per cent., although in trichinosis they may amount to as much as 60 per cent. of the leucocytes. Increase of the eosinophile percentage is known as "*eosinophilia.*"

The eosinophiles are diminished during the course of non-febrile tuberculosis, and in croupous pneumonia, many infectious diseases running a high temperature. In the latter disease a return to the normal proportion is regarded as a warning of the approach of the crisis.

(*f*) **Basophilic Leucocytes; Mast Cells.**—These cells are found in but very small percentage in the healthy blood, not amounting to more than 0.5 per cent. of the total leucocytes. They possess a polymorphous nucleus. Their granulations require the following for their staining:

Filtered alcoholic saturated solution of dahlia,	50.00 c.c.
Glacial acetic acid,	10.00 c.c.
Distilled water,	100.00 c.c.

They range in size from 20 μ to 30 μ. Their clinical significance does not seem to have been fully settled; but they are found in greatly-increased proportion—as much as 10 per cent.—in splenic leukæmia.

(*g*) **Myelocytes.**—These are never present in normal blood. They are of two varieties, according to the staining qualities of their granules, which may be neutrophilic or eosinophilic.

The **neutrophilic myelocytes** present a large, round, feebly-stained nucleus, herein resembling the large mononuclear lymphocytes, but differing from them in their possession of neutrophilic granules. They range in size from 12 μ to 20 μ. They are regarded as transition forms between the large mononuclear leucocytes and the polymorphonuclear neutrophiles. They are found in large numbers in the blood of splenic leukæmia; but they may also occur in chlorosis, pernicious anæmia, and some leucocytoses.

The **eosinophilic myelocytes** resemble the preceding in all particulars excepting in the staining qualities of their granules, which are eosinophilic. They are found in association with the neutrophilic myelocytes, though in much smaller numbers.

The Number of Leucocytes.—The number of leucocytes in normal blood is stated to vary from 5000 to 7000 per cubic millimetre. In proportion to the number of red corpuscles present, their number is 1 to 500 to 1 to 600.

Physiological Leucocytosis.—An increase in the number of leucocytes is called leucocytosis or hyperleucocytosis. The number of leucocytes in a cubic millimetre of blood sufficiently large to be called leucocytosis has been arbitrarily placed at 10,000. Leucocytosis may be physiological or pathological. Physiologically, leucocytosis may result from

a. Digestion (digestive leucocytosis).
b. Pregnancy.
c. Infancy.

(*a*) **Digestive Leucocytosis.**—An increase in the number of leucocytes begins within an hour after a full meal, especially after one consisting very largely of nitrogenous food. It reaches its maximum during the period of intestinal digestion, at which time the leucocytes may be, in proportion to the red corpuscles, as 1 to 150. The increase relates almost exclusively to the polymorphonuclear neutrophiles. Exceptionally, the relative proportions of the several varieties of leucocytes are retained.

While the occurrence of digestive leucocytosis may be utilized for diagnostic purposes, its value is impaired by the fact that it is sometimes absent in perfectly healthy persons; and, as the result of disease, it is not always affected in the same manner. It is most pronounced in persons of good digestive powers. It is absent in the majority of cases of gastric cancer. It has, therefore, been suggested as a sign for the differentiation of that affection and gastric ulcer, digestive leucocytosis not being impaired in the latter.

(*b*) **Leucocytosis of Pregnancy.**—Leucocytosis in pregnancy is common, but not invariable. The number of leucocytes never goes above 14,000 per cubic millimetre. It is observed with especial frequency and to a most pronounced degree during the last three months of pregnancy and in primipara. It reaches its maximum at the time of delivery, and then gradually declines. The increase relates almost exclusively to the polymorphonuclear neutrophiles.

(*c*) **Leucocytosis of Infancy.**—The number of leucocytes at birth is usually from 10,000 to 20,000, and continues so for the first two days. Then it begins to decline until, at the end of the first week, it has reached the normal for infancy (10,000). The increase relates to the polymorphonuclear neutrophiles, which may constitute 60 to 70 per cent. of the whole, as compared with the normal of 40 per cent. for the infant.

Pathological Leucocytosis.—The pathological conditions which may be characterized by leucocytosis include the following:

a. Inflammations and infections (inflammatory leucocytosis).
b. Leucocytosis of malignant disease.
c. Post-hæmorrhagic leucocytosis.
d. Toxic leucocytosis.
e. Leukæmia (leucocythæmia).

(*a*) **Inflammatory Leucocytosis.**—Leucocytosis occurring during the course of inflammatory and infectious disorders is to be regarded as an effort on the part of the system to overcome the invading principle, be it bacterial or otherwise. While the study of such a leucocytosis, when taken in conjunction with the associated symptoms, is capable of giving considerable information, of itself, it is valueless clinically. Its extent bears no relation to the severity of the inflammatory process, but rather to the degree of intensity of the infection and the reacting powers of the individual. It is more marked in inflammations with purulent than in those with serous exudates.

To determine, then, the full significance of the leucocytosis, it is necessary to be possessed of full information concerning the case. Thus, a slight increase would indicate either mild infection or feebly-reacting powers; which, the associated symptoms will decide.

(*b*) **Leucocytosis of Malignant Disease.**—The leucocytosis is by no means of constant occurrence in malignant disease, being observed in a little less than half the cases. Even then it is almost always moderate in degree. Exceptions to this latter statement are found in some rapidly-growing tumors of the kidneys, lungs and liver, in which the leucocytes may reach as high as 50,000. To reconcile these discrepancies, Da Costa states the general rule that "tumors of rapid development, involving a large area of tissue and complicated by extensive metastases, cause decided, often high, leucocytoses; while localized tumors of small size and slow growth give rise to trifling, if any, increase." The increase relates in most instances to the polymorphonuclear neutrophiles; exceptionally, to the lymphocytes, especially in sarcoma of the lymphatic system.

(*c*) **Post-Hæmorrhagic Leucocytosis.**—Leucocytosis follows very promptly upon severe losses of blood, *i.e.*, at intervals ranging from two to ten hours. It is especially well-marked after traumatic hæmorrhages, rather than those resulting from lesions in which the general strength and reactive powers of the patient are impaired. The leucocytosis usually runs less than 25,000, and disappears, as a general rule, in about three or four days. The polymorphonuclear neutrophiles are the elements usually increased. Exceptionally, there is a lymphocytosis. Myelocytes are frequently present.

(*d*) **Toxic Leucocytosis.**—The toxic conditions of especial interest capable of producing leucocytosis include coal-gas and ptomaine poisoning, ether narcosis, cholæmia, and uræmia.

(*e*) **Leukæmia; Leucocythæmia.**—In leukæmia, of which there are two varieties, the spleno-medullary and the lymphatic, the number of white cells is enormously increased,—to 100,000 or more. The blood-changes in these are best studied in connection with the other clinical features of the disease.

Hypoleucocytosis; Leucopenia.—By leucopenia is meant a diminution in the number of white blood-cells. It may be either physiological or pathological.

Physiological leucopenia may follow exposure to cold, brief hot baths, or stimulation of sensory nerves. It is a phenomenon of starvation and malnutrition.

Pathological leucopenia may be observed in a variety of infectious diseases when the infection is great and the resisting powers of the individual are weak. Among the special diseases of the blood it is occasionally observed in chlorosis, and is rather common in pernicious anæmia, but in neither of these affections is it a diagnostic symptom. It has also been known to follow the ingestion of certain drugs, notably ergot, sulphonal, tannic acid, atropine, agaricine, and picrotoxine.

8. **The Parasites of the Blood.**—The principal parasites found in the blood include

1. The malarial parasite.
2. Filaria sanguinis hominis.
3. Schistosoma-hæmatobium.
4. Spirillum obermeieri.

1. **The Malarial Parasite; The Method of Examination.**—The malarial parasites may be discovered and studied from either fresh or dried and stained specimens of blood. The former is, beyond all question, the best adapted to clinical work, as it may be carried out at the bedside. The essential points in the preparation of the specimen are cleanliness of the slide and cover-glass, and the depositing on the former of a very small portion of blood. If too much blood is taken, the corpuscles will be in layers, and will form rouleaux. Thus their edges only will be visible, and the discovery of the various forms, the pigment-granules excepted, will be impossible.

If it is not practicable to make the examination at once, the blood may be deposited on a warm slide and the cover-slide sealed with vaseline. These precautions will enable the parasites to retain their vitality for a couple of hours.

When it is desired to examine dried and stained specimens, the film is prepared in the usual way, after which it is fixed by ether and alcohol. It is then stained with Plehn's solution, the formula of which is as follows :

Concentrated aqueous solution of methylene blue, . .	60 c.c.
One-half per cent. solution of eosin in 75 per cent. alcohol, .	20 c.c.
Distilled water, ...	40 c.c.
Twenty per cent. solution of sodium hydrate, . . .	12 gtt.

After fixation, the films are subjected to this solution for five or six minutes. As a result, the malarial organisms are stained blue ; the nuclei of the red or white corpuscles, blue or violet ; large granulations, red.

Wet specimens may be stained by puncturing the skin through a drop of a solution of methylene blue in fresh ascitic fluid.

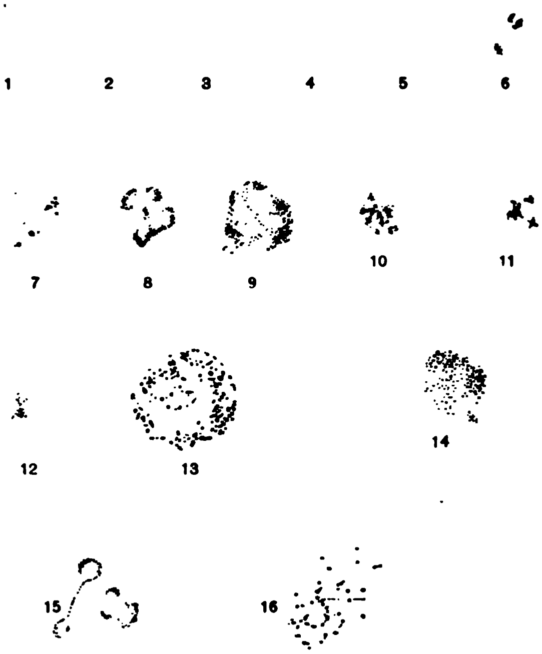

FIG. 166.—THE TERTIAN PARASITE.

1. *Normal erythrocyte.*
2, 3, 4, 5. *Intracellular hyaline forms.*
6, 7. *Young pigmented intracellular forms.* In 6 two distinct parasites inhabit the erythrocyte, the larger one being actively ameboid, as evidenced by the long tentacular process trailing from the main body of the organism. This ameboid tendency is still better illustrated in 7, by the ribbon-like design formed by the parasite. Note the delicacy of the pigment granules, and their tendency toward peripheral arrangement in 6, 7, and 8.
8. *Later developmental stage of 7.* In 7, 8, and 9 enlargement and pallor of the infected erythrocyte become conspicuous.
9. *Mature intracellular pigmented parasite.*
10, 11, 12. *Segmenting forms.* In 10 is shown the early stage of sporulation—the development of radial striations and peripheral indentations coincidentally with the swarming of the pigment toward the center of the parasite. The completion of this process is illustrated by 11 and 12.
13. *Large swollen extracellular form.* Note the coarse fused blocks of pigment. (Compare size with that of normal erythrocyte, 1.)
14. *Flagellate form.*
15. *Shrunken and fragmenting extracellular forms.*
16. *Vacuolation of an extracellular form.*

NOTE.—The original water-color drawings were made from fresh blood specimens, a Leitz $\frac{1}{12}$-inch oil-immersion objective and 4 ocular, with a Zeiss camera-lucida, being used.

(From DaCosta's "Hematology".)

The usual errors, as a result of which failure to discover the parasite ensues, are the taking of too large a specimen of blood, so that nothing is visible under the microscope but numerous rouleaux formations ; not taking sufficient time to the examination ; and making examinations after quinine has been administered.

As already stated, the examination of fresh specimens is the most satisfactory.

It is a misconception that the malarial parasites exist in such numbers that one has but to peer through the microscope to see them in droves all over the field. As a matter of fact, but one or two organisms are visible on each field ; and, in very many instances, one must search through a number of fields before discovering even one. The observer should never, therefore, give up his search for them until he has spent a half-hour at the work.

If the examination is made after quinine has been administered, no micro-organisms whatever may be discoverable.

The novice to whom the appearance of the parasites is strange should direct his attention to the discovery of black specks possessed with motion. These are the pigment-granules. The hyaline forms appear as clear areas in the midst of unusually pale-red cells ; they are irregular in shape and endowed with amœboid motion. In certain cells the parasite itself may not be visible, but its presence is suggested by their extra large size. The detection of the flagellæ is generally simplified by their active movements, which cause a commotion among the surrounding red cells, the movements of which suggest a search for the cause of this activity and result in the discovery of the flagellæ.

Three varieties of the malarial parasite have been described, namely,

a. The tertian parasite.
b. The quartan parasite.
c. The æstivo-autumnal parasite.

Each of these gives rise to its own peculiar type of malarial fever (see p. 63).

A thorough conception of the appearance of the malarial parasite demands, first, a knowledge that it passes through several stages in its course of development, each stage presenting well-defined characteristics. These stages include the following :

i. The intracellular hyaline forms.
ii. The intracellular pigmented forms.
iii. The extracellular pigmented forms.
iv. The segmenting forms.
v. The flagellate forms.

(*a*) **The Tertian Parasite.**—i. THE INTRACELLULAR HYALINE FORMS.—These constitute the first stage in the development of the tertian parasite. They are visible in the blood immediately or shortly following the paroxysm. They are characterized by small transparent (hyaline) bodies in the red blood-cells. They possess active amœboid movements; hence they exhibit frequent and varied changes in shape. These hyaline bodies are derived from the sporulation of the previously maturing set of parasites. Next we have

ii. THE INTRACELLULAR PIGMENTED FORMS, which appear in about twenty-four hours after the paroxysm, and consist of a mass of pigment granules, yellowish-brown in color, deposited (at first scantily) about the peripheral portion of the parasite. Like the hyaline bodies, these granules are possessed of an active amœboid motion.

With progressive development, the parasite increases in size, the pigmentation grows more dense, and the amœboid movements gradually disappear. While all this is taking place, the containing corpuscle becomes paler and larger, until finally it is decolorized, and its substance forms a mere rim about the organism. It may happen, however, that with the development of the pigmentation the parasite escapes from its host, and then we have

iii. THE EXTRACELLULAR PIGMENTED FORMS.—The organisms undergoing this accident are immediately dwarfed and deprived of their amœboid motion. They may become fragmented. Parasites pursuing this course are soon lost to view by reason of obscuration of their outlines.

If, however, the parasite does not escape from the corpuscle until approaching the stage of segmentation, it then appears in the blood as a large spherical body filled with actively-moving pigment-granules, some massed in groups and figures, others separate and distinct. It is from these extracellular pigmented forms that the flagellated bodies are derived.

The stage of development succeeding the intracellular pigmented forms is

iv. THE SEGMENTING FORMS.—These are observed in greater numbers in the deep circulation,—only occasionally in the blood taken from the finger-tip. The first stage of segmentation is characterized by the collection of the pigment-granules into a compact mass at or near the centre of the parasite. Then appear, serially, a number of minute refractive points and indistinct parallel linear shadings, and indentations are observed where the latter intersect the edge of the parasite. Thus the latter is divided into some fifteen or twenty segments, which are the spores from which the next succeeding group of parasites is developed. Each spore contains a central refractive spot. At this stage the organism is enclosed by a thin layer of erythrocyte, which, however, is scarcely visible by reason of its transparency. Now the corpuscle ruptures, and the spores or segments escape into the surrounding blood, and disappear from view.

v. FLAGELLATE FORMS.—These, as already stated, develop from the

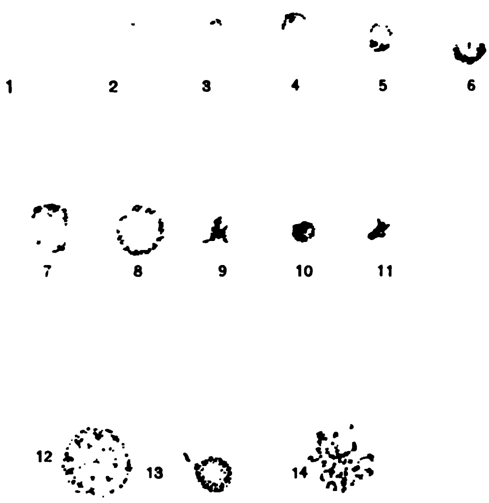

FIG. 167.—THE QUARTAN PARASITE.

1. *Normal erythrocyte.*
2. *Intracellular hyaline form.*
3. *Young pigmented intracellular form.* Note the coarseness, dark color, and scantiness of the pigment granules.
4, 5, 6, 7. *Later developmental stages of 3.* Note the peripheral distribution of the pigment in all the parasites from 3 to 8.

8. *Mature intracellular form.* Note that the stroma of the erythrocyte is no longer demonstrable.
9, 10, 11. *Segmenting forms.* In 9 are shown the characteristic radiating lines of pigment.

12. *Large swollen extracellular form.*
13. *Flagellate form.*
14. *Vacuolation of an extracellular form.*

(From DaCosta's "Hematology".)

extracellular pigmented bodies which have escaped from the erythrocytes just before the time for segmentation. The appearance of flagellation is preceded by great activity of the movements of pigment-granules, which finally accumulate at or near the centre of the parasite, and one or more long, tentacular arms are thrust forth from the main body. These flagella exhibit one or more bulbous enlargements, situated usually at their distal extremity, less frequently in their continuity or at their central terminus. These flagella are now believed to be true male sexual organs. They disappear in one of two ways, namely, by detachment from the main body, and are lost in the blood-stream; or they gradually disappear, apparently by reabsorption.

(*b*) **The Quartan Parasite.**—It is generally admitted that the distinctions between the hyaline forms of the quartan parasite and those of the tertian are so poorly defined that they are hardly sufficient for a satisfactory differentiation of the two organisms, excepting in the hands of the expert. The quartan bodies are smaller, less actively motile, and more refractive than the tertian.

The pigmented forms present features which are readily recognized. In the first place, their outlines are far more distinct than those of the tertian. The pigment-granules themselves are dark-brown, almost black, and early in their course become collected in one portion of the organism. The amœboid movements of the parasite become entirely lost at the end of forty-eight hours.

The corpuscle itself presents a marked contrast with the condition observed in the tertian variety. It is shrunken, dark, or what has been described as "brassy."

The process of segmentation involves a division into six to twelve segments, with the pigment-granules collected in the centre.

(*c*) **The Æstivo-Autumnal Parasite.**—Marked irregularity characterizes the phenomena of the æstivo-autumnal fevers, and this has been explained by the irregularity as to the time required for its parasites to complete their growth. There are observers who claim that the æstivo-autumnal fever is produced by several organisms, concerning some of which we have no knowledge.

The hyaline forms of the æstivo-autumnal parasite are much smaller than those of either the tertian or quartan varieties. Its margins are much more sharply delimited. Its characteristic feature, by which it may be recognized with certainty, lies in the formation of ring-like bodies. After a time, by reason of the amœboid movements, it changes into a flattened disk. Later it returns to the ring-like form. And so the changes recur.

The pigmented forms appear as a few brown granules, situated usually towards the edge of the parasite. They are generally non-motile.

Parasites in a more mature stage of development are to be found only in the deeper circulation. In order to study them, it is essential to aspirate an internal organ, preferably the spleen. The segmenting forms may thus be studied. They may make regular rosettes with eighteen or twenty segments, or they may be very irregular. About their centre, the pigment-granules are massed.

The erythrocytes containing the organism early show degenerative changes, as a "brassy" appearance of their protoplasm with crenation of their edges.

The characteristic crescents, ovoid and round bodies, are not found in the peripheral blood until a week or more after infection. They are liable to be found in the blood for some time after the subsidence of all symptoms, and are exceedingly resistant to the action of quinine. These bodies are believed to be transformations of such of the intracellular hyaline forms as have not undergone the process of segmentation.

2. **The Filaria Sanguinis Hominis.**—The embryo of the filaria sanguinis hominis appear in the human blood as small worms, measuring $\frac{1}{75}$ of an inch in length by $\frac{1}{8000}$ of an inch in thickness. They are readily visible under a low-power ($\frac{1}{5}$) objective. They are found in the peripheral blood only during the resting-hours of the patient. The number varies all the way from five or ten to over two thousand per cubic millimetre of blood. They are actively motile, their movements consisting of wriggling and twisting, but they make but little shifting of location in these movements.

In many cases the parasite produces no disturbance in health whatever, being discovered by accident when the patient is subjected to a thorough examination. Sometimes they produce obstruction of the lymphatics, resulting in chyluria, lymphangitis, lymph scrotum, and elephantiasis.

3. **Schistosoma Hæmatobium.**—This is a parasite which infests the smaller veins of the portal system, producing cystitis and hæmaturia, sometimes associated with anæmia. They never occur in the peripheral blood. They are discoverable only in the blood-clots discharged from the bladder.

4. **Spirillum Obermeieri.**—This is the specific element of relapsing fever, and is found in the blood of patients suffering from that disease during the stage of pyrexia. It is a thin, curled, fibre-like body, $\frac{1}{500}$ to $\frac{1}{8000}$ of an inch in length.

The Widal Reaction.—The Widal reaction is based upon the property possessed by blood from typhoid patients of causing clumping of the typhoid bacilli in young cultures in bouillon. It is now universally regarded as very valuable evidence of the presence of that disease, although it is occasionally absent in cases of typhoid fever, and very exceptionally, for some unexplainable reason, appears in other conditions, or in healthy sub-

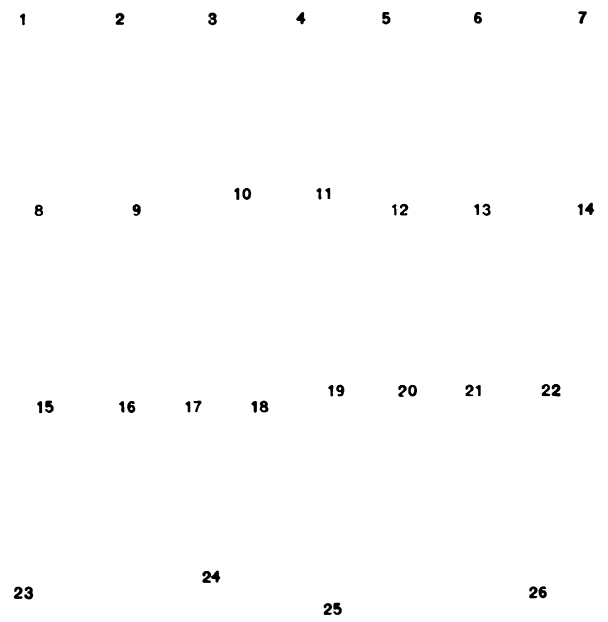

FIG. 168.—THE ESTIVO-AUTUMNAL PARASITE.

1. *Normal erythrocyte.*
2, 3. *Young hyaline ring-forms.*
4, 5, 6. *Intracellular hyaline forms.* In 4 the parasite appears as an irregularly shaped disc with a thinned-out central area. In 5 and 6 its ameboid properties are obvious.
7. *Young pigmented intracellular form.* Note the extreme delicacy and small number of the pigment granules.
8, 9. *Later developmental stages of 7.*
10, 11, 12. *Segmenting forms.*
13, 14. *Crescentic forms at early stages of their development.*
15, 16, 17, 18, 19. *Crescentic forms.* In 15 and 19 a distinct "bib" of the erythrocyte is visible. Vacuolation of a crescent is shown in 18, and polar arrangement of the pigment in 17.
20. *Oval form.*
21, 22. *Spherical forms.*
23. *Flagellate form.*
24. *Vacuolation and deformity of a spherical form.*
25. *Vacuolated leucocyte apparently enclosing a dwarfed and shrunken crescent.*
26. *Remains of a shrunken spherical form.*

(From DaCosta's "Hematology".)

Young cultures of the typhoid bacilli are absolutely essential to the proper performance of the test. They must not be more than twenty-four hours old. To prepare such a culture, some of the micro-organisms from an agar culture should be transplanted to bouillon.

There are three methods of applying the Widal reaction, as follows:

a. The dry method.
b. The fresh-blood method.
c. The separated serum.

In each of these methods it is necessary that the specimen be sufficiently diluted with typhoid bouillon, never less than in the proportion of one to ten; and some observers habitually use much higher dilutions, as one to fifty. In well-marked cases, the reaction is obtainable in the proportion of one to two hundred.

(*a*) **The dried-blood method** is one most commonly in use, because of the ease with which the blood-specimen may be prepared for transportation to laboratories at a distance. It is the one almost universally employed by Boards of Health. A few drops of blood from the suspected case are deposited upon a piece of glazed non-absorbent paper, or upon a glass slide, and permitted to dry. In this condition it will keep for many days. To make the test, if the specimen is on a glass slide, it is moistened with a drop of sterile water and thoroughly mixed, with the aid of a platinum loop. Next, it is diluted with nine drops of typhoid bouillon culture. If, however, the specimen is on paper, the portion covered by the blood is carefully cut out with a pair of scissors, and soaked thoroughly, face downwards, in ten drops of bouillon culture. The specimen is then ready for examination.

(*b*) **The Fresh-Blood Method.**—In this method the fresh blood is mixed at the patient's bedside with ten times its bulk of typhoid bouillon in a test-tube. It is then ready for examination, which must be made within two or three hours after the preparation of the specimen. Accurate dilution may be secured by specially constructed pipettes, or by the Thoma-Zeiss leucocytometer.

(*c*) **The Serum Method.**—To obtain the specimen of serum, a quantity of blood (15 to 20 drops) is collected in a test-tube, and allowed to coagulate. The fluid serum is then drained off, and is ready for examination as soon as it has been diluted with ten times its bulk of typhoid bouillon.

To make the examination, a drop of the mixture of blood and bouillon is placed upon a cover-glass and then placed face downward upon the concave surface of a specially constructed glass slide. The specimen is then examined under the microscope as a hanging drop. Some observers are satisfied to use an ordinary flat slide.

The *reaction* which takes place in the presence of typhoid serum is as follows: The bacilli, at first actively motile, are soon attracted to each other, and become clumped in large masses; hence the term "*agglutination reaction*." This change must take place promptly, *i.e.*, within from a few seconds to thirty minutes. The subsequent course of the reaction varies, and possesses no clinical significance. The masses may undergo granular degeneration, and finally disappear; they may remain unaltered; or they may break up after a few hours and their places be supplied by a few actively motile organisms.

In all cases, the culture itself should be examined before each test is made, as small clumps of bacilli are of occasional occurrence. In the absence of this precaution, such clumping may lead to erroneous conclusions.

Symptomatology and Clinical Course of the Important Diseases of the Blood.

1. Chlorosis.

The etiology of chlorosis is sufficiently obscure to make it an unimportant factor in the diagnosis of the affection. As a matter of fact, the vast majority of cases occur in young women, and this is always to be regarded as suggestive. Nevertheless, the disease may occur in males and in older subjects. The first thing to impress the observer is the extreme pallor of the patient,—a pallor that has been described as possessing a greenish-yellow color; hence the common name of the disease, "green sickness." Sometimes the patient's face flushes readily, giving rosy cheeks on a pale background. The mucous membranes, as far as they may be visible, are likewise pale.

This extreme pallor is dependent upon characteristic changes in the blood. To the naked eye that tissue is thin, pale and watery-looking. Examination shows it to be remarkably deficient in hæmoglobin, even to the extent of 20 per cent. or less of the normal proportion.

The red corpuscles likewise decrease in number, though never to the extent of the hæmoglobin. In the milder cases they may be fully up to the normal standard. Even in the more severe examples of the disease they do not go below 3,000,000 per cubic millimetre, and in no case reported have they gone below 1,300,000 (Hayem's case). So far as the erythrocytes present are concerned, many of them are undersized (microcytes). In the most severe cases macrocytes may be observed. Poikilocytes may also occur, though they are rare. The general nutrition of the patient is apparently good, because of the good deposit of fat; nevertheless, such patients have soft and flabby muscles, and are easily tired and exhausted. Sustained effort of mind or body is impossible, and they complain of shortness of breath, palpitation, and vertigo. Digestive disturbances, including defective or perverted appetite, discomfort after eating, and constipation, are commonly present. Heart-murmurs, systolic in time in the mitral, tricuspid and pulmonary areas, are often heard. Those in the mitral and tricuspid areas are dependent upon relative insufficiency; the systolic pulmonic murmur is the characteristic murmur of anæmia. There may also be heard, in the majority of cases, a venous hum (*bruit de diable*) over the jugular at the base of the neck. There is some degree of dilatation of both ventricles, particularly the left. The heart-action is irritable, palpitation ensuing upon slight cause. The pulse is of low tension. Utero-ovarian symptoms are common, but they are the result, and not the cause, of the disease. The most important of them is the amenorrhœa, which may be very persistent.

The prognosis of chlorosis is good when proper treatment is instituted. If permitted to go untreated for too long a period, complications may appear. Among these are ulcer of the stomach, venous thromboses, and endocarditis. Intercurrent acute diseases are not borne as readily as by the healthy.

2. Pernicious Anæmia.

The etiology of pernicious anæmia is shrouded in obscurity. Hunter has lately, with some good reason, endeavored to show that it is dependent upon infection introduced by way of the mouth (oral sepsis.)

The patient presents a highly anæmic appearance, but the complexion is peculiar in that it is of a lemon-yellow or canary tint. At times this tint may be so marked as to simulate jaundice. It is dependent upon the presence of a pigment derived from the destruction of blood-cells. The profound anæmia brings with it proportionately severe exhaustion. Emaciation is not likely to be present, but the muscular tissue is fat and flabby. The diagnosis, however, rests upon an examination of the blood. Macroscopically, it is thin and of watery appearance. At times it is difficult to obtain a sufficient quantity from the puncture to fill the stem of the cytometer. The number of red-blood cells is greatly diminished. Counts of 1,000,000 are common, and others in which the number is less than 500,000 to the cubic millimetre are not infrequent. The hæmoglobin percentage is likewise reduced, but usually not to the extent suggested by the low blood-count. The conclusion is, therefore, that the individual richness of the remaining red blood-corpuscles in hæmoglobin is not impaired; on the contrary, it may be increased. Very characteristic are the appearances of the individual blood-elements under the microscope. Poikilocytes are numerous; megalocytes, megaloblasts, microcytes; in fact, these changes may be summed up as great variations in shape and size of the erythrocytes. The megaloblasts, in particular, are regarded as especially significant from a diagnostic standpoint. The hæmoglobin in many of the erythrocytes is very irregularly distributed, and this gives rise, in unstained specimens, to the impression that they are nucleated. Such cells stain irregularly. The leucocytes are generally diminished in number, but there is a relative lymphocytosis.

The usual circulatory symptoms attendant upon anæmia, as palpitation, shortness of breath, soft pulse, venous hum, and cardiac murmurs, are common. Hæmorrhages are not unusual, and are observed in the retina with especial frequency. Epistaxis, hæmatemesis, hæmoptysis, enterorrhagia, menorrhagia and ecchymoses may also occur. Indeed, they may be mistaken for the primary affection, and lead to incorrect treatment.

Remarkable among the symptoms of pernicious anæmia is the fever, which at times may reach a high grade. This fever is believed to be due to the presence of a poison in the blood. The urine is usually normal in appearance, but contains, in some cases, a large amount of pathological urobilin. Various digestive disturbances may be encountered. Some cases are apparently associated with an atrophy of the gastric mucous membrane, and this fact has been utilized to explain the genesis of such instances.

Of late years, frequent attention has been directed to the supervention of symptoms pointing to spinal-cord lesions. As a rule, the general features of these cases bear a close resemblance to those of combined sclerosis of the posterior and lateral columns (ataxic paraplegia). But pure tabes dorsalis and primary lateral sclerosis have also been simulated.

The prognosis of pernicious anæmia is invariably unfavorable. Cases usually run an irregular course, improving to a remarkable extent for a time, but ultimately succumb to the disease.

The diagnostic errors relate mainly to cases in which early hæmorrhages are believed to be due to local lesions, and assigned as the cause of the anæmia, and treated accordingly. If attention is paid to the microscopic examination of the blood, it seems incredible that it can be mistaken for the other forms of anæmia, or for the anæmia attendant upon certain organic diseases, as Bright's disease and cancer.

3. Leucocythæmia; Leukæmia.

Two varieties of leukæmia are now recognized, namely, the **spleno-medullary** and the **lymphatic**. While each of these affections possesses definite clinical features, by which they may be readily recognized, nevertheless, their differentiation should always be based upon the results of examination of the blood.

Spleno-Medullary Leukæmia.—This variety of leukæmia is invariably insidious in its onset and slow in progress. Its course, however, is interrupted by periods of remission and exacerbation of symptoms. In the beginning the patient exhibits the usual concomitants of anæmia, as pallor, prostration, breathlessness, etc. He early complains of a sense of fullness or weight, and sometimes pain, in the region of the spleen. These symptoms receive a ready explanation in the greatly-increased size of the spleen and the distention of its capsule. Sometimes the pain is due to congestion of that organ or to perisplenitis. Sometimes the patient complains of tenderness of various bones, especially, however, the sternum and tibiæ. In outward appearance the patient presents one important distinction from the anæmias thus far described, in that he is usually emaciated, sometimes to a remarkable extent.

Perhaps the most prominent symptom, aside from the anæmia, is the enlargement of the spleen, which may be associated with some tenderness. The enlargement usually attains great proportions, but it is very apt to vary in size from time to time.

The circulatory symptoms include well-marked weakness of the heart, palpitation, breathlessness on slight exertion, the venous hum at the root of the jugular, and mitral, tricuspid and pulmonic systolic murmurs. Hæmorrhages are by no means uncommon, and may proceed from any organ or any part of the surface of the body. Especially common are epistaxis, bleeding from the gums, hæmatemesis, enterorrhagia, and retinal hæmorrhages.

Pigmented patches and petechiæ are sometimes observed on the skin. The former, however, may be due to the extravagant use of arsenic as a therapeutic agent.

Some cases approach the lymphatic form of leukæmia, and present some degree of enlargement of the lymphatic glands.

As already stated, the examination of the blood furnishes the only reliable information upon which to base a diagnosis. The first striking feature is the greatly-increased number of leucocytes, which may exist in proportion to the erythrocytes as 1 to 6 or 8, and, in extreme cases, as 1 to 1. This disproportion depends not only upon absolute increase in the number of leucocytes, but also upon diminution of the red blood-corpuscles. The former not infrequently amount to 300,000 per cubic millimetre, and extreme cases in which the count amounted to 1,000,000 have been recorded. The deficiency in red corpuscles is not usually extreme, the count ranging from 2,000,000 to 4,000,000 per cubic millimetre. The hæmoglobin percentage is generally disproportionately low to the erythrocyte count.

The leucocyte count presents some remarkable fluctuations within a short time. According to Hayem, there may be variations of 100,000 between morning and evening. In view of these remarkable fluctuations, it is possible to overlook the existence of a leukæmia by reason of the good condition of the blood at the time of making the examination.

The special blood symptom which points unequivocally to splenomedullary leukæmia is the presence of a *large* number of myelocytes. Under no circumstances should they be less than 20 per cent. of the total leukocytes, and they may amount to 50 per cent. Many of these myelocytes are of unusually large size; others are dwarfed and distorted and small, and therefore atypical.

Other changes in the leucocytes may be observed, but the above are the only ones which we have come to regard as characteristic of spleno-medullary leukæmia.

Lymphatic Leukæmia.—The lymphatic form of leukæmia differs from the spleno-medullary variety especially in the predominance of glandular enlargements and the small size of the splenic enlargement. It is, however, to the blood, as already stated, we look for a differentiation. The leucocytes are increased in number in lymphatic leukæmia, but it is in exceptional cases only that the count goes above 100,000 to the cubic millimetre. This increase relates almost entirely to the lymphocytes, which constitute about 90 per cent. of the total leucocytes present.

While lymphatic leukæmia usually pursues a chronic course, exceptional cases have been recorded in which the disease tends rapidly to a fatal issue.

4. Splenic Anæmia.

Splenic anæmia is a form of profound anæmia characterized by great enlargement of the spleen without lymphatic enlargements or increase in the number of leucocytes. In its early stage it presents the ordinary features of anæmia, without any sign or symptom by which its nature may be recognized. Later, however, the enlargement of the spleen appears, and is associated with considerable pain in the left hypochondrium. In the meantime the anæmia, and with it the strength of the individual, becomes progressively worse. Hæmorrhages from different portions of the body constitute an important symptom. Fever of remittent type appears, and may create the impression that the anæmia is due to a septic process. There is, however, little or no leucocytosis. The erythrocytes are greatly diminished in number, sometimes to one-fourth the normal standard. The hæmoglobin percentage is disproportionately low to the erythrocyte count.

5. Secondary Anæmia.

The term "secondary anæmia" is applied to those cases of anæmia which result from a determinable cause in distinction from those occurring otherwise, and which are typified in pernicious anæmia, leucocythæmia and chlorosis. Secondary anæmia may be acute or chronic. The acute cases occur mostly after hæmorrhage. They may also result from septic infection and certain poisons, notably chlorate of potash and the snake poisons. Such cases present no diagnostic difficulties, excepting in cases of concealed hæmorrhage.

Chronic symptomatic anæmia may result from repeated hæmorrhages; chronic metallic poisoning, as from lead and arsenic; various toxæmia, notably that accompanying renal disease; diseases characterized by the drain of albuminous constituents of the blood, as prolonged suppuration, chronic dysentery, phthisis, lactation, and dropsy; unsanitary surroundings, including bad air and unwholesome food; infectious diseases, especially malaria, syphilis, typhoid fever and rheumatic fever; diseases characterized by malnutrition, as those of the digestive apparatus, and malignant disease; and, lastly, intestinal parasites.

(*a*) **Post-Hæmorrhagic Anæmia.**—The constitutional symptoms are profound, owing to the rapidity with which the loss of blood takes place. The amount of blood the loss of which is capable of producing marked systemic disturbance is sometimes surprisingly small, as little as 40 c.c. causing changes in the quality of the blood, and larger quantities distinct symptoms. The erythrocytes diminish in number for several days after loss of blood; hæmoglobin percentage also decreases, but with rapidity disproportionate to the corpuscular loss. With improvement in the condition of the blood, the red cells increase in number much faster than the restoration of hæmoglobin percentage. The special clinical feature of stained blood-specimens is found in the large number of normoblasts present. Minor changes observed include slight poikilocytosis, leucocytosis, the increase relating to the polymorphonuclear neutrophile. Very exceptionally the increased leucocyte count may depend upon a lymphocytosis.

(*b*) **Anæmia from Septicæmia.**—Septic processes are ofttimes associated with anæmia, sometimes of profound character and rapid onset. Cases have been reported in which the blood-count fell to 1,500,000 within a few days.

The chronic symptomatic anæmias depend for their diagnosis mainly upon the presence of symptoms pointing to some primary lesion capable of causing the blood deterioration, and the absence of the blood-changes and associated signs of the essential anæmias. The constitutional manifestations of the anæmia itself differ in no particular from those attending other anæmias.

The examination of the blood develops some interesting phenomena. The puncture sometimes fails to cause the flow of a sufficient quantity for a full examination, owing to the reduction in the quantity present in the system. Hæmoglobin and the erythrocyte count, especially, however, the former, are reduced. Stained specimens show irregular staining of the red blood-cells, poikilocytosis, nucleated red corpuscles, leucocytosis relating especially to increase of the polymorphonuclear neutrophiles, and, in the cases dependent upon intestinal parasites, eosinophilia.

6. Anæmia Infantum Pseudo-Leukæmia.

This is an affection first described by von Jaksch, and defined by him as follows: "There is often marked swelling of the spleen, the liver, and the glands, with continued and decided leucocytosis. The relation of the white corpuscles to the red may be 1 to 20, 1 to 17, or 1 to 12, but, despite these features, there is no evidence of leukæmia at the post-mortem examination."

It is a matter of observation that nearly all cases occur prior to the age of four years, and the majority of these between the seventh and twelfth months. Cases have been observed in connection with rachitis, hereditary syphilis, and gastro-intestinal disturbances, especially chronic diarrhœa.

The pseudo-leukæmia of infancy is of gradual onset, the initial manifestations consisting of weakness and pallor. Aside from the usual phenomena of anæmia in general, there are presented enlargement with hardness of the spleen, sometimes associated with increased size of the liver. Careful examination of the lymphatic glands will show these also to be enlarged, but never to the extent of forming distinct visible tumors. The blood-changes, however, are such as to suggest a secondary anæmia. They include marked diminution in the red corpuscles, sometimes to as low as 1,000,000 per cubic millimetre; increased leucocyte count to from 30,000 to 50,000 per cubic millimetre. The increase relates mainly to the polymorphonuclear neutrophiles, though sometimes to the lymphocytes.

The main difficulty in diagnosis rests in the differentiation from leucocythæmia. The blood examination decides the nature of the case, the increased leucocyte count in the disease under consideration being due to increase of the polymorphonuclear neutrophiles. Leukæmia, moreover, does not present the enlargement of the liver, or a history of rachitis or syphilis. The pseudo-leukæmia of infancy does not necessarily follow a fatal course.

7. Hæmophilia.

Little or nothing can be said concerning the etiology of hæmophilia other than its tendency to occur in families. It is transmitted by atavism by the female members, but males are more frequently affected than females. The existence of the disease is not manifested until some small cut or injury, as the withdrawal of a tooth, has been followed by persistent or severe hæmorrhage. Sometimes the bleeding appears spontaneously, and may be manifested as epistaxis and extravasations into the connective tissues and joints. Of special importance in the diagnosis of the affection is the involvement of the joints, especially of the elbows and knees. This accident is usually associated with fever, swelling and pain, and may be followed by organic changes.

A diagnosis of hæmophilia should not be based upon a single persistent oozing of blood following a slight wound. It is necessary that the clinical picture of heredity and joint-affections be associated. Examination of the coagulability of the blood demonstrates that it takes from eight to fifteen times the normal required.

8. Scurvy; Scorbutus.

Notwithstanding the extensive observations made of this disease, absolute certainty respecting its origin has not been attained. While there is a growing tendency to attribute it to a specific micro-organism, the trend of opinion still favors the long-held idea that it is due to the depreciation of those elements of nutrition which can only be supplied by fresh vegetables as part of the diet. Some claim that it is dependent upon toxic materials in the food, and there is much to support this view.

The onset of the disease is almost invariably slow and insidious. The initial symptoms are not at all diagnostic, consisting, as they do, of a slowly-increasing weakness, with anæmia. The skin soon presents changes, such as dryness and roughness, with a dirty or pale-yellow staining. Blotches resembling the marks produced by bruises appear (petechiæ). The characteristic lesions, however, appear in the mouth, and consist of swelling and sponginess of the gums, which bleed on slight manipulation. This symptom, however, is not likely to appear in edentulous subjects. It is especially marked about the necks of defective or diseased teeth. Of almost equally great diagnostic importance are the subcutaneous swellings. They are most numerous about the ankles and calves, but may appear on any part of the body; their margins are not well defined; they are tender on manipulation.

With the further progress of the disease, which, in the present day of medical knowledge, is not liable to occur, the weakness becomes more profound; respiration is weak and labored; the patient suffers from palpitation; the heart-muscles become weak. General emaciation, with œdema of the lower extremities, appears. The cutaneous ecchymoses become progressively more prominent. The swelling of the gums increases until these structures appear like vascular growths, which may eventually break down and form ulcers. Advanced cases of scurvy exhibit a remarkable tendency to sanguineous extravasations, especially beneath the periosteum and into the serous cavities. As complications, pleurisy, pneumonia, gangrene of the lungs, pericarditis with sanguinolent effusion, diarrhœa, dropsy and hydrarthrosis may appear.

With such characteristic symptoms as above outlined, the diagnosis of scurvy should not be difficult; and yet the disease is frequently overlooked. When it appears in a number of individuals living under identical unhygienic conditions, the nature of the cases is at once recognized. But scurvy may appear in single individuals as the result of personal peculiarities. The victim may have been unable to procure suitable food by reason of poverty; he may have been following some dietetic scheme with more zeal than judgment when under treatment for some other disorder. In any case, multiplicity of lesions serves as a strongly-suggestive sign of the disease.

Of late years considerable attention has been paid to *infantile scurvy*, or *Barlow's disease*. Its signs and symptoms are such as to make its diagnosis easy, and yet the disease is frequently overlooked. The origin of infantile scurvy is generally too much persistence in artificial foods without the administration of a suitable quantity of fresh milk. It is not necessary, in the production of the disease in infants, that the food be one of the proprietary articles, for the worst case I ever examined resulted from the persistent use of sterilized milk, the case being regarded by previous attendants as one of congenital syphilis.

The symptoms appear somewhat more rapidly than in the case of adults. There is, however, a period of anæmia and debility preceding the appearance of the characteristic symptoms. The little one first exhibits undue irritability and uneasiness, and then,—and this is very characteristic,—exhibits a remarkable intolerance of being handled. The legs in particular are tender on any manipulation. The pains are aggravated by the slightest movement; but the child is fairly comfortable while quiet. Very shortly there will appear fusiform swellings of the thighs, due to subperiosteal effusion ; the same condition may involve the tibiæ. The upper extremities are involved less frequently. The bone involvement is especially liable to cause changes in the epiphyses, leading to their separation, as shown by the appearance of crepitus. While all of these changes are going on, the anæmia and weakness progress, the skin assumes the appearance already described in adult scurvy, and the gums swell and become spongy. The teeth in relation to the affected gums become loosened and may fall out. The hæmorrhagic tendency observed in adult scurvy is present in the infantile case and is evidenced principally by subcutaneous extravasations, though also by epistaxis and intestinal hæmorrhage. Cases of infantile scurvy are liable to be mistaken for paralysis, rachitis, or rheumatism. Analysis of the symptoms, and a comparison of the symptoms presented with those of the affections with which it may be confounded, should make a diagnosis certain, especially when the method by which the patient has been fed is known. Notwithstanding the severity of the symptoms, proper treatment usually brings about prompt recovery in cases of scurvy.

9. Purpura.

Strictly speaking, purpura is but a symptom. The many conditions in which it may be present is well illustrated by the statistics of the London Hospital, 200 cases observed in that institution being dependent upon no less than 23 different affections. Hæmorrhagic effusions, associated with evidences of anæmia, are essential features of purpura. The classifications of the varieties of the disease are as varied as the authors are many. Stephen Mackenzie arranges the pathological conditions under which purpura may occur as follows :

"I. (a) The infective diseases, in nearly all of which, but especially in small-pox, measles, scarlet fever, cerebro-spinal fever, syphilis and malaria, purpura is an occasional incident.

"(b) Rheumatism.

"(c) The various conditions under which certain organic matters present in excess in the blood,—such as bile, urinary constituents, or certain adventitious organic poisons, such as snake-venom,—may gain access to the blood.

"(d) The presence in the blood of extraneous chemical substances, such as phosphorus, mercury, mineral acids, salicylic acid, iodide of potassium.

"(e) Conditions in which some constituent of the blood is wanting, as in scurvy.

"(f) Alterations in the formed elements of the blood, as in anæmia and leucocythæmia.

"To series I., the term 'vascular purpura' may be given.

"II. Mechanical Purpura. (a) Conditions that offer an impediment to the circulation, general or local; as in diseases of the heart and large blood-vessels and tumors compressing them, thrombosis, temporary vascular spasm, or paralysis; as in convulsive seizures, whooping-cough, or angina pectoris.

"(b) Want of mechanical support of blood-vessels, as in wasting in the newly-born and the aged.

"III. Conditions in which the direct influence of the nervous system can be traced, as in tabes, neuralgia, and the like. To this series the name 'nervous purpura' may be applied.

"IV. Congenital imperfection of the blood-vessels, as hæmophilia—'hæmophilic purpura.'"*

With such a diversity of conditions capable of presenting purpura as a manifestation, it is manifestly impossible to assign it any definite clinical course. We can only say that most of the diseases presenting purpura present also changes in the blood, fever, albuminuria, gastric disturbances, and hæmorrhages.

Certain varieties of purpura are worthy of special mention, as follows:

Purpura Simplex.—The characteristic feature of this variety of purpura is the outbreak of hæmorrhagic extravasations on various portions of the cutaneous surface. If constitutional symptoms are associated they are but slight, and consist, at the most, of slight fever, general indisposition, and disordered digestion. The hæmorrhagic spots may come out in crops. After a few days they cease to appear. The lesions gradually fade away, leaving some cutaneous pigmentations to mark the site of the hæmorrhages for some little time.

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* Allbutt's "Practice of Medicine," vol. v., p. 571.

Purpura Hæmorrhagica; Morbus Werlhofii.—This represents a much more serious condition than the preceding. It is characterized by the cutaneous extravasations as in purpura simplex; but there are also hæmorrhages from the various mucous membranes, of which those from the nose, mouth, throat, stomach, lungs, and urinary organs are the most common. The constitutional disturbance is much more marked, including fever, headaches, vomiting, and epigastric pain. The subcutaneous hæmorrhages are ofttimes quite large, and may be attended by well-defined œdema of the surrounding tissues. The associated anæmia sometimes reaches a high grade, erythrocyte counts as low as between one and two million having been reported. Purpura hæmorrhagica is a serious disease. Some cases present a foudroyant course, coming on suddenly and terminating fatally within a few days (*Purpura fulminans*). Recovery in the favorable cases is a tedious matter, extending as it does over a number of weeks, the hæmorrhages gradually ceasing. The patient is left, at the best, highly anæmic.

Purpura Rheumatica; Peliosis Rheumatica; Schönlein's Disease.—The majority of cases of purpura rheumatica occur in persons between the ages of ten and forty years. It seems to bear a definite relation to rheumatism, and occurs under the same climatic conditions as those favoring that disease. After a prodromal period of indefinite duration, the arthritic symptoms appear. These consist of pain, heat, and swelling in joints, notably in those of the ankles and knees. The shifting tendency of the lesions observed in rheumatism does not occur in this disease. The purpuric eruption appears at about the same time as the joint-lesions. It consists of numerous petechiæ, which may congregate in patches. The more dependent portions of the body seem to be more liable than those above. Hæmorrhages from the mucous membranes are rare, but have taken place from the mouth and throat. More or less well-marked œdema of the feet and ankles is almost invariably present. The pain and stiffness are much worse in the after-parts of the day. Constitutional disturbance is nearly always manifested, as fever, anæmia and prostration. The prognosis of peliosis rheumatica is, as a rule, favorable. The clinical course of the disease is, however, very variable.

Henoch's disease; Henoch's purpura is characterized by purpuric extravasations and joint-lesions, in association with severe abdominal symptoms, as pain, vomiting and diarrhœa. The urine is usually albuminous, and under the microscope is shown to contain casts and blood-cells. The disease is a serious one, death resulting from nephritis or by exhaustion after a long illness.

CHAPTER XVII.

SYMPTOMS RELATING TO THE EXTERNAL SURFACE, INVOLVING, FOR THE MOST PART, ALTERATIONS IN SHAPE AND SIZE.

Changes in shape and size of the body as the result of disease may be either general or local. The former class of cases includes general emaciation and obesity. The latter term is usually employed to designate changes limited to a particular part or member of the body; although it may, with equal exactness, be applied to atrophies or hypertrophies of particular tissues, *e.g.*, the muscles.

Emaciation.—The term "emaciation," as ordinarily employed, suggests diminution in the size of the body as the result of inanition or malnutrition. It is first manifested by diminution and final disappearance of the subcutaneous fat, followed in order by wasting of the muscles, liver, bones, and heart. It is readily recognized by simple inspection, when the observer is acquainted with the appearance of the individual in health. For purposes of accuracy, however, it should be estimated by weighing the patient. It is always accompanied by loss of strength, which is in direct proportion to the loss of flesh.

Emaciation is, however, a comparative term, indicating, as it does, a departure of a particular individual from the normal in a certain direction. We are accustomed to rely upon a fixed relative proportion between the weight of an individual and his height, usually stated as ranging from twenty-five to thirty pounds for each foot in stature. This, however, is an arbitrary standard for very general guidance only, considerable departures from which are not inconsistent with perfect health. Hutchinson has endeavored to give us a more exact standard, and has formulated the following:

A man of 4 ft. 6 in. to 5 ft. 0 in. ought to weigh about 92.26 lbs.
5 ft. 0 in. to 5 ft. 1 in. ought to weigh about 115.52 lbs.
5 ft. 2 in. to 5 ft. 3 in. ought to weigh about 127.86 lbs.
5 ft. 4 in. to 5 ft. 5 in. ought to weigh about 139.17 lbs.
5 ft. 6 in. to 5 ft. 7 in. ought to weigh about 144.29 lbs.
5 ft. 8 in. to 5 ft. 9 in. ought to weigh about 157.76 lbs.
5 ft. 10 in. to 5 ft. 11 in. ought to weigh about 170.86 lbs.
5 ft. 11 in. to 6 ft. 0 in. ought to weigh about 177.25 lbs.

This table shows that with increase in height a greater proportion of weight to each inch must be allowed. In judging of the relation of weight to condition of health, one must not adhere too strictly to an arbitrary ideal, but consider the following points before deciding that a departure from the normal exists: 1. The habit of the individual. 2. The size of the skeleton. 3. The development of muscular tissue. 4. The development of adipose tissue and its distribution.

The term "habit" is used in the above in a double sense, viz., to refer to the natural build of the individual,—as to whether he is stout, spare or thin by nature,—and to refer to his habits as to exercise, etc. "Stout" would imply a simple increase of adipose tissue, evenly distributed over the entire body. "Spare habit" designates a build of body with but little adipose, but with bones and muscles well put together. Such individuals may also be said to be "wiry." They are usually possessed of good powers of endurance of fatigue and resistance to disease. Persons given to athletic sports are apt to exhibit but little subcutaneous fat.

The weight of an individual is not always to be gauged by his size or girth, for it must be remembered that bone and muscle add more, relatively, to it than does adipose tissue. Hence the size of the bony framework and the size of his muscles must always be taken into consideration.

A consideration of other questions suggested by the preceding paragraphs will be deferred until we study "Obesity." (*Vide* page 633.)

It may be stated in a general way that emaciation attends all pathological processes in which nutrition is depraved, or in which the normal balance between waste and repair is disturbed. We observe it in many acute diseases, especially those characterized by high and prolonged fever. The duration of the fever, however, is more effective in bringing about emaciation than is its severity. Acute diseases attended by profuse discharges, such as cholera Asiatica, are attended by remarkable loss of weight, —due, however, not to loss of adipose, but to dehydration. This is exhibited by the patient in a pinched expression of countenance and a shrivelling of the skin over various portions of the body. Loss of weight from this cause is regained very rapidly after the source of dehydration has been abolished. For example, in a case of toxic gastro-enteritis the patient lost seven pounds within twelve hours by reason of excessive vomiting and purging; but as soon as he was in condition to take food and drink, the weight was regained as rapidly as it was lost.

The majority of chronic maladies other than those of the nervous system are attended by emaciation. In some of them the loss of flesh is extreme. This last remark applies especially to organic lesions of the digestive system (organic dyspepsias), pulmonary tuberculosis, and malignant disease. In digestive disturbances it is the result of a lack of nutritive elements furnished to the body. In pulmonary disease it is due to a combination of causes, namely, attendant gastro-intestinal disease, lack of appetite, constitutional disturbance, and prolonged suppuration. The wasting is first noticed about the chest, in this class of cases. In malignant disease, on the other hand, emaciation first shows itself about the face. It does not always make itself noticeable until the disease is well advanced, *i.e.*, has progressed to the stage of ulceration, or is interfering in some way with the processes of nutrition.

Diabetes is, in the majority of instances, accompanied by remarkable loss of flesh, and this seems to have a direct relation to the severity of the glycosuria. At the same time it must be borne in mind that the gastro-intestinal disorders usually attendant upon diabetes, and the restricted diet, play some part in its production.

Very interesting is the remarkable degree of emaciation ofttimes attendant upon chronic pancreatic disease. While sometimes due to disturbed digestion, this cannot always be the case, for generally the starches, etc., are acted upon by other digestive processes than those dependent upon the pancreas. It has been suggested by Harley, and with good reason, that it is the result of an auto-intoxication resulting from the cessation of the functions of the pancreas as an organ of internal secretion.

Emaciation in children, occurring as the main symptom, is to be regarded as indisputable evidence of improper feeding or of some chronic constitutional disease. As to the first mentioned, it must be remembered that infants may emaciate when the proper food is given in excessive quantities as well as when food is deficient in quality or quantity, or both. Among the chronic disorders of infancy producing emaciation, those most frequently observed are congenital syphilis, rachitis and tuberculosis.

Loss of flesh is not a prominent factor in the majority of cases of nervous and mental disease. When present in hysteria, it is never as marked as the apparent severity of the other symptoms would lead us to expect. Very rarely we observe cases of persistent hysterical vomiting in which emaciation and prostration are profound, such cases being known as anorexia nervosa.

Certain nervous diseases are attended by marked wasting of the muscular tissues. A consideration of these will be found in the chapters relating to the nervous system.

Emaciation in the insane is due, as a rule, to the habits of the individual as regards the taking of food and physical activity rather than to the disease *per se.* It is to be especially noted in acute mania, acute delirium, and in delusional insanities in which fear of poisons constitutes a prominent feature.

Obesity.—Obesity, being the result of the taking of nutriment in excess of that required for repair, is rarely to be regarded as a pathological condition. In the majority of cases it is dependent upon physiological causes, which include

1. Hereditary tendency.
2. Sedentary, easy-going, lazy habits.

3. Age.
4. Occupation.
5. Diet.
6. Climate.

Undue accumulation of adipose, even though the result of physiological causes, must eventually become a source of annoyance to the individual, and also be productive of secondary changes in important viscera.

1. **Heredity.**—It is a well-known fact that individuals of a family are apt to exhibit similar tendencies as to build, though there are noticeable exceptions to this observation. Sometimes the tendency to obesity is transmitted as the only peculiarity; in others it is associated with evidences pointing to such constitutional vices as rheumatism, gout, lithæmia, oxaluria, and cholelithiasis.

2. **Habits.**—Persons of easy-going, lazy habits, are inclined to take on an undue amount of adipose because they are generally fond of the pleasures of the table, and because they do not take sufficient exercise to oxidize even a normal amount of food. Their self-satisfied state of mind contributes also to this result. Conversely, we note that active persons, and those of worrying, restless disposition, are apt to be spare of build.

3. **Age.**—The normal infant is expected to have an undue amount of fat. Women at or after the climacteric age are inclined to become unduly stout, partly because of physical inactivity and improper feeding, and partly because of the changes in the sexual apparatus. The same remark applies, though with less force, to men at or beyond middle life.

4. **Occupation.**—Occupation as a cause of obesity is closely related to the amount of physical exercise permitted.

5. **Food and Drink.**—Excessive feeding and drinking, especially the taking of starches, sugars and fats, and the drinking of alcoholic beverages, incline to obesity. Personal idiosyncrasy is, of course, an important factor, for we have all seen persons of thin, spare habit, who indulge freely in food and drink, and very stout women who have very poor appetites. The form in which alcohol is taken is undoubtedly an important factor; the rotund form of the regular beer drinker is well-known. Accumulation of fat from alcohol addiction always presents a flabby consistence, lacking the firmness of normal adipose.

6. **Climate, Including Race.**—Obesity, of course, is observed in every clime, and among all conditions of people. At the same time, it is observed with especial frequency among the inhabitants of damp, low-lying countries, as witness the well-known corpulence of the Dutch. The racial tendency of the Hottentots to obesity and of the Hindus to spareness is equally well known.

The normal proportion of fat to the entire body is generally estimated at one-twentieth of the bodily weight in men, and slightly more than that amount in women. The fat formation is found mostly in the subcutaneous, subserous and intermuscular connective tissue. It is especially prevalent about the abdomen, buttocks, breasts and neck, while it is absent, or nearly so, from the wrists, ankles, eyelids, scrotum and penis. It is never seen beneath the peritoneal coat of the stomach or intestines, while it exists in large quantity in the great omentum. Ordinarily, the omental fat amounts to but one-half pound. It may be increased, however, to seven or eight pounds, and one case has been reported in which it amounted to thirty pounds.

As the result of disease, accumulation of fat is a comparatively infrequent phenomenon; but when it does occur, it is, as in the case of the physiological laying on of adipose, an expression of delayed or deficient oxidation, as in the anæmias and certain pulmonary and cardiac diseases, and after hæmorrhage and mercurial and arsenical poisoning.

Obesity, even though it results from perfectly physiological causes, must be regarded as a disease. To use the words of Hippocrates, "Corpulence is not only a disease itself, but is the harbinger of others." The physical activity of the patient is nearly always diminished, owing to increased weight, and at the same time the normal strength is wanting, because of fatty infiltration of the muscular tissues. Excretion is active, as observed in the excessive sweating. Free drinking of water is resorted to, to compensate for the dehydration thus occasioned. Obesity, then, carries with it the very causes which lead to its increase, namely, physical inactivity and free indulgence in liquids.

The corpulent should not be regarded as robust. They are apt to fall ill periodically, without apparent cause. They are liable to respiratory catarrhs and diarrhœa, probably because the self-regulation of temperature is impaired by the thick layer of subcutaneous fat and the weakening of the circulation by fatty infiltration of the heart-muscle. When suffering from acute febrile maladies, their disease must be viewed with considerable concern, because of diminished heat radiation and the enfeebled heart.

Dropsy.—By dropsy is meant an accumulation of serum in the connective-tissue spaces or in one of the serous cavities of the body. When the serous accumulation is limited to the connective tissue of some particular portion of the body, we are apt to refer to it as œdema. When it is widespread or general, we call it anasarca. When involving a serous cavity, it is given special names according to its location, *e.g.*, ascites, dropsy of the peritoneal cavity; hydrothorax, when involving the pleural cavity; hydrocephalus, when involving the cranial cavity; hydrarthrosis, when there is effusion into the synovial sac of the joints; hydropericardium, when in the pericardial cavity, etc.

General factors in the production of dropsy: Dropsy depends upon the exudation of a greater amount of serum into the tissues than can be taken up by the absorbents. This, in turn, may result from

(*a*) Increased exudation, absorption being normal.
(*b*) Deficient absorption, exudation being normal.

Increased exudation may result from one of a variety of factors, as follows:

(*a*) Alterations in the calibre of the arteries and capillaries.
(*b*) Changes in the structure of the vascular walls.
(*c*) Changes in the composition of the blood.

Diminished absorption is due to vascular obstruction, which may be either

(*a*) Lymphatic, or
(*b*) Venous;

and this vascular obstruction may be either

(*a*) General, or
(*b*) Local.

The general conditions leading to the production of œdema are especially apt to be present in connection with diseases of

(*a*) The heart,
(*b*) The kidneys, or
(*c*) The liver.

It is seldom, indeed, that any one of the above-tabulated agencies in the production of dropsy exists alone in any given case. Alterations in blood-pressure are apt to co-exist with changes in the walls of the vessels and alterations in the quality of the blood.

Increased exudation is observed in connection with dilatation of the arteries more frequently than from any other cause. The œdema accompanying local inflammation is the result of arterial dilatation, although it is due in part to increased permeability of the vascular walls. Dilatation of the vessels with its consequent œdema may also result from vaso-motor paralysis. A good example of this is seen after section of the sciatic nerve of the rabbit after it has been joined by its sympathetic fibres. Vaso-motor paralysis is one factor in the production of the dropsy of chlorosis and other forms of anæmia, the other being alteration in the composition of the blood.

Alterations in the composition of the blood *per se*, it is claimed, cannot produce œdema. It is essential that they be associated with changes in the vascular walls before increased transudation of serum is possible. As already stated, hydræmia is a factor in the production of the dropsy of chlorosis and other forms of anæmia, including that appearing after frequently repeated losses of blood. It also plays an important part in the œdema of renal diseases. The hydræmia here is produced by the large loss of albumin and the diminished excretion of water by the kidneys.

Vascular degenerations are, however, almost invariably present, as are also changes in blood-pressure.

Alterations in the walls of the capillaries, leading to greater permeability, are probably factors in all cases of œdema. There is no doubt that this condition exists in all renal diseases, and in most advanced cases of cardiac trouble. Without it, it is hardly probable that the associated dropsies could be of the high grade so frequently observed. As already stated, it is an important factor in the production of inflammatory œdema.

Absorption takes place mainly through the veins and lymphatics, mainly the former. Obstruction of either of these vessels leads to œdema of the parts drained by the obstructed vessels. Owing, however, to the very free anastomoses of the lymphatics, it is very rarely that œdema is occasioned by any interference with their functions. When, however, the larger lymphatic trunks, especially the thoracic duct, are obstructed, œdema may result. This is observed in the chylous ascites due to obstruction of the thoracic duct, and in the œdema of elephantiasis.

The best illustration of a dropsy arising from venous obstruction is seen in the ascites observed in connection with such liver diseases as are associated with pressure upon the portal vein, *e.g.*, cirrhosis of the liver. Other notable examples are œdema of one leg from phlegmasia alba dolens (milk-leg) as a sequel of confinement or typhoid fever, and œdema of the left leg from cancer of the sigmoid flexure.

The anasarca accompanying organic heart affections is due to general venous stasis. It can only occur in connection with failure of the cardiac muscle, with or without valvular disease. The first step in the production of the dropsy is a damming back of blood in the passive congestion of the lungs; and this is followed by a backing up of blood in the right ventricle, and thence into the auricle. Then the circulation through the systemic veins is impeded and transudation of serum takes place, favored, moreover, by alterations in the structure of the vascular walls.

Symptoms and Clinical Relations of Œdema.—Œdematous infiltrations may be of either slow or rapid onset, usually the former. When involving the lymph-spaces of the connective tissue, as in the dropsy of heart disease, it tends to seek the most dependent parts; hence it is most apt to be observed first in the feet of persons who are about, and along the inner and posterior surface of the thighs and over the sacrum in those confined to bed. A notable exception to this statement is the œdema of renal disease, which nearly always makes its first appearance about the eyelids. As the exudation increases, there is an enlargement of the affected part in all directions; depressions and cavities are obliterated, and the normal contour of the extremity is lost. Pressure over the swollen tissues produces a pitting, which remains for some little time after the pressure has been removed. This symptom of œdema—"pitting on pressure"—is to be observed in the early stages of œdema, especially over the tibia. As the transudation continues, the swelling extends higher and higher up the legs, and at the same time the distention of the parts becomes so great as to cause them to become quite hard. At this time the skin is pale, and its temperature subnormal. The skin may be unable to resist the pressure within, in which case it ruptures, and we observe oozing of serum from numerous small openings. This state of affairs is always a most unfortunate one, for dermatitis, gangrenous processes, ulcerations, and other serious cutaneous lesions result. If the œdema is continued over a great length of time, secondary changes may take place. These are believed to be due to defective nutrition from compression and from the circulation of toxic substances in the blood, leading to a hyperplasia of the connective tissue. After paracentesis, the skin also is found to be thickened and adherent to the structures beneath.

Dropsical fluid is watery in character, and has a specific gravity ranging from 1006 to 1016, and is comparatively poor in corpuscles and albuminous constituents. When the transudation has been due in great part to alterations in the vascular walls, as in inflammation, the fluid is more dense, and contains relatively more corpuscles and albuminous substance.

Local œdema is due, as a rule, to a local cause, *i.e.*, to obstruction of the vein draining the œdematous area.

Œdema of an arm may result from enlargement of the axillary lymphatic glands.

Œdema of the arms, or of the arms, head and thorax, suggests an intrathoracic tumor, *e.g.*, aneurysm or enlarged mediastinal glands. The obstruction is in the superior vena cava. If that obstruction is above the entrance of the azygos vein, the œdema will not invade the chest.

Suddenly-appearing œdema of the arms and head suggests that very rare condition, aneurysm rupturing into the superior vena cava.

Œdema of the feet is, in the majority of cases, the starting-point of a general dropsy, partly because the feet are the most dependent parts of the body, and partly because the circulation there is feeble, being the farthest removed from the heart. It may also occur in persons of relaxed fibre after prolonged exertion,—a long day's work. Such an œdema may continue for years without other evidence of impaired health. Œdema of the legs and wrists is sometimes observed during convalescence from infectious fevers, and is indicative of great weakness.

Œdema of the eyelids, as already stated, is usually the beginning of renal dropsy. At first it is observed only in the morning; in advanced cases it persists throughout the day. The very loose character of the cellular tissue about the eyes makes this part the seat of infiltrations which are not of serious significance. For example, dermatitis about the face is not infrequently associated with marked œdema of the eyelids. This same symptom may be due to bites of insects, urticaria, etc. Œdema of the eyelids may occur as a pure neurosis, especially in children at about the age of puberty.

Localized œdemas are sometimes symptomatic of deep-seated inflammation or vascular obstruction. Œdema over the mastoid may be due to mastoiditis or thrombosis of the lateral sinus; over the parotid gland, to parotitis; over the ribs, to empyema; over the præcordia, to suppurative pericarditis; over the liver, to hepatic abscess or suppurative cholecystitis; over the abdomen, to suppurative peritonitis, etc.

Diagnosis of Œdema.—Œdema may be mistaken for

(*a*) Inflammatory swelling.
(*b*) Myxœdema.
(*c*) Connective-tissue dystrophies.
(*d*) Emphysematous swelling.

Inflammatory swelling is accompanied by the classical symptoms of inflammation, pain, heat and redness.

The **myxœdematous swelling** does not pit on pressure; it is widely diffused; there is atrophy of the thyroid gland, and numerous nervous phenomena characteristic of that condition. (*Vide* page 640.)

Connective-tissue dystrophies are hard, localized swellings, which do not pit on pressure, and are situated irrespective of the dependent position of the parts.

Emphysematous swelling can only occur in connection with an injury to the respiratory tract permitting of the escape of air into the connective-tissue spaces. The swollen parts do not pit on pressure, and palpation produces a crackling sensation.

Certain Rare and Unusual Forms of Œdema.—Angioneurotic Œdema.—Cases coming under this class differ greatly in clinical features. They are all due to vaso-motor disturbance, and they furthermore agree in having a rapid onset, and in attacking neurotic subjects. Usually the œdema involves parts about the face rather than elsewhere. Still it may affect any part, whether submucous or subcutaneous. The œdema may be either circumscribed or diffuse. It has been known to settle in the tissue back of the eyeball, producing exophthalmos. In two cases reported by Osler the larynx was the point of attack, and a fatal issue ensued. Most of the cases of œdema occurring in women during or about the menstrual periods belong to the class under discussion.

Rheumatic œdema occurs in the subjects of acute rheumatism, and in persons of rheumatic diathesis. It occurs under such variable conditions that its diagnosis is always a matter of extreme difficulty.

Malarial œdema is apparently a sub-variety of angioneurotic œdema, depending upon malarial infection. The cases thus far reported in literature present no special features, so far as the œdema itself is concerned, but are characterized by the peculiar periodical recurrence belonging to malarial diseases, and by their prompt cure under anti-malarial medication.

Myxœdema.—The swelling of myxœdema is generally distributed. It is due to the infiltration of a mucoid substance into the connective tissue, and has atrophy of the thyroid gland as its primary cause. Unlike the dropsy, the swelling does not tend to invade the most dependent parts. Indeed, so far as the extremities are concerned, the reverse condition seems to obtain, for the arms are more swollen than the hands, and the legs more than the feet. The swollen parts do not pit on pressure. The skin is dry and scaly. This symptom is an important one, but is greatly neglected by clinicians. The patient is apt to speak of the large accumulation of scales which she can shake out of her stockings each evening. The appearance of the face is characteristic. It is generally swollen and rounded, and has been described as "moon-shaped." Glistening, pear-shaped swellings are sometimes to be seen below the eyes. The nostrils are broad and thickened. The lips are thickened and everted. The tongue is involved in the swelling, and this, in conjunction with the mental hebetude characteristic of the disease, makes the speech slow and labored. The hands and feet are enlarged, the former being described as spade-like. The bodily temperature is subnormal. Women are the main subjects of the disease. Amenorrhœa is always a prominent symptom; indeed, it may for a time be the one to which most attention is paid.

Emphysema.—This is a condition characterized by the presence of air in the connective-tissue spaces. It can only occur when there is a wound of one of the air-containing viscera, *e.g.*, the respiratory organs or the intestines; usually, however, the former. The swelling has a local beginning, *i.e.*, near the injured organ, and may spread rapidly, and invade the entire body. The swollen tissues sink readily under the examining fingers, but do not pit on pressure. If it is not extensive, it may be driven from place to place by manipulation. Palpation gives a peculiar crackling crepitus; percussion a superficial resonance. It results usually from wounds of the lungs, fractured ribs, and rupture of air-vesicles by violent expiratory efforts, as in coughing, straining at stool, or during parturition.

The air may escape first into the interlobular connective tissue, and thence into the subcutaneous tissues. In these cases there is a suddenly-appearing dyspnœa, and it may even be that the patient dies very shortly.

Localized emphysema of the face suggests a fracture involving the antrum.

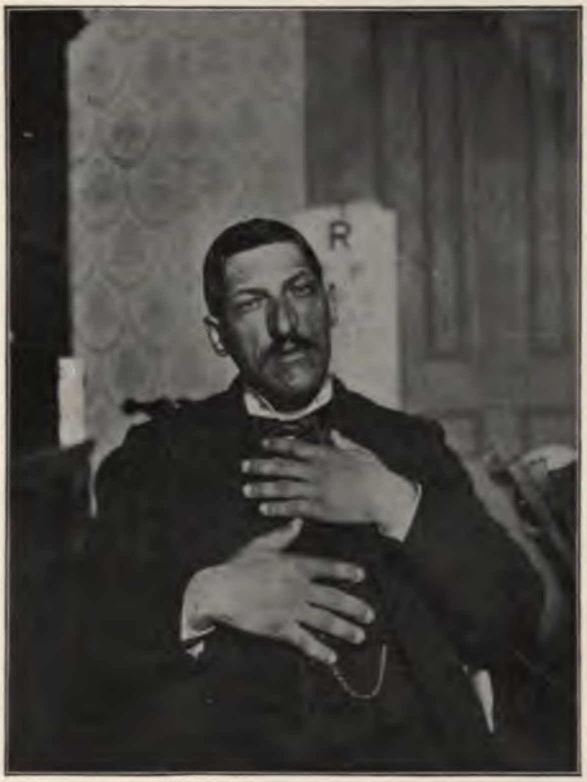
Emphysema of the flanks occurs as a symptom of rupture of the lower third of the duodenum.

Emphysema of the perineum and scrotum has occurred in connection with wounds of the lower bowels.

Emphysema occasionally occurs as a result of rapidly-spreading moist gangrene, being due to the accumulation of gases formed by the resulting decomposition.

Acromegaly.—Acromegaly is characterized by enlargements involving bony structures as well as soft parts, and widely distributed throughout the body. The parts affected are especially the bony prominences, the bones of the hands and feet, superior and inferior maxillæ, the sternum, the pelvis, and the spinal column. The cartilages of the ears, nose and larynx may also participate in the pathological process. The appearance of the face, hands and feet, and the curvature of the spine, constitute the main factors on which the diagnosis is based. The general contour of the head may be described as an oval, the face being disproportionately large, as compared with the cranium. The superciliary ridges are enlarged and prominent, and this gives the forehead a retreating aspect. The bony prominences, especially the occipital protuberance and the malar processes, are greatly enlarged. The nose is hypertrophied in all its parts, making it an altogether too prominent feature. The changes in the inferior maxilla are

FIG. 169.

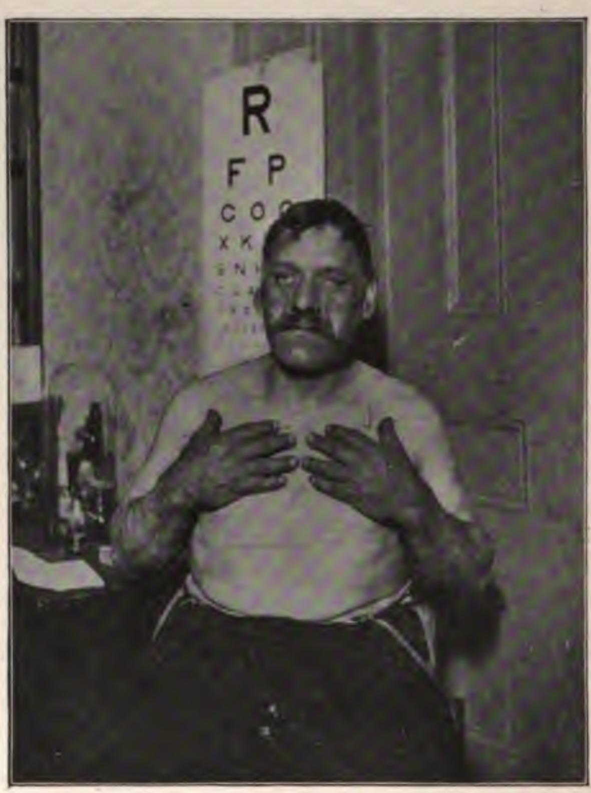


Case of acromegaly. Mr. X.

especially characteristic, although not invariably present. This bone is enlarged in all its parts, but especially so in its body. The angles of junction between rami and body are more obtuse than normal, and the anterior extremity of the bone projects beyond the upper jaw, producing the condition known as prognathism.

The carpal and metacarpal bones are enlarged in thickness, but not in length. The soft parts surrounding them are somewhat thickened. These changes give the fingers a shape which has not inaptly been described as that of a sausage. The thickening of the tissues about the palms cause characteristic appearances here also. The same changes are observed in the feet. Changes in the spinal column and ribs lead to dorsal kyphosis; sometimes to scoliosis and a compensatory dorso-lumbar lordosis.

FIG. 170.



Case of acromegaly. Mr. Z.

The tissue-changes in acromegalia are practically always symmetrical.

Associated with the objective conditions above described there may exist hæmianopsia, severe headaches, amenorrhœa, loss of strength, diminished sexual power, and a host of symptoms, which, however, are not of much value from a diagnostic standpoint. Their presence often tends to render the true nature of the case obscure, as, causing the main suffering, they take attention from the cardinal symptoms of the disease.

Osteo-Arthropathie Hypertrophiante Pneumique.—This is a condition first described by Marie in 1890. The enlargement is limited to the joints, and is associated with pulmonary diseases, as empyema, bronchitis, and tumors. The finger-, elbow-, shoulder-, toe- and knee-joints may all be affected. The fingers are enlarged, but the main portion of the hands is uninvolved. The soft parts are normal. The finger-nails have the appearance of being too large for the fingers, and are spread out at the sides and curved over the ends of these members. The ends of the fingers themselves are enlarged and bulbous, and have not inaptly been compared to the end of a drumstick. The feet and toes exhibit changes corresponding to those observed in the hands.

Osteitis Deformans.—A proper understanding of the symptoms of osteitis deformans necessitates a brief review of the pathology of the disease. It is a widely-diffused osteitis, involving mainly the compact structure of the bones, which become thickened, soft, spongy, and vascular. There is an excessive production of imperfectly-developed granulation tissue.

Osteitis deformans rarely occurs before the age of fifty, and never before forty. The long bones of the extremities become misshapen and curved from the superincumbent weight of the body. The hands and feet are rarely involved. The cranial bones undergo enormous thickening, while those of the face remain normal. This gives the head a triangular shape, with the base directed upwards. The attitude of the body also demands attention. It is decidedly stooped, with rounded shoulders, bowed and rigid spine, and chin overhanging the sternum. This condition gives the body a shortened appearance, which is made all the more apparent by comparison with the changes in the long bones of the extremities. In many cases, especially in their early stages, pains unassociated with constitutional symptoms are prominent phenomena.

Alterations in the Shape and Size of the Head.

The alterations which may take place in the shape and size of the head depend upon pressure from within, the formation of inflammatory exudates or tumors without, traumatism, or errors in development, and are as follows:

Enlargements:
  a. Hydrocephalus.
  b. Rachitis.
  c. Cretinism.
  d. Syphilis.

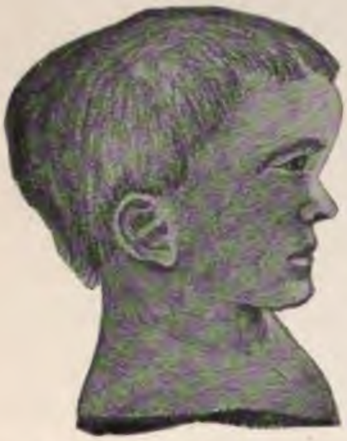
Diminution :
- a. Microcephalus.

Alteration in shape :
- a. Asymmetry.
- b. Swellings of variable origin.
- c. Depressions, which, in turn, may be due to
  - i. Fractures.
  - ii. Adherent cicatrices.
  - iii. Maldevelopment and malnutrition of bone structure.

The enlargements and diminutions above tabulated, being dependent upon definite pathological changes, present individual characteristics making their recognition easy. These peculiarities may be epitomized as follows :

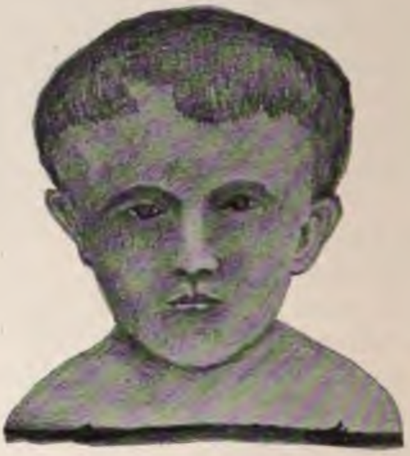
An enlarged head approaching a square shape indicates rachitis. A similar condition is sometimes observed as one of the phenomena of hereditary syphilis.

FIG. 171.



Hydrocephalic head—side view.

FIG. 172.



Hydrocephalic head—front view.

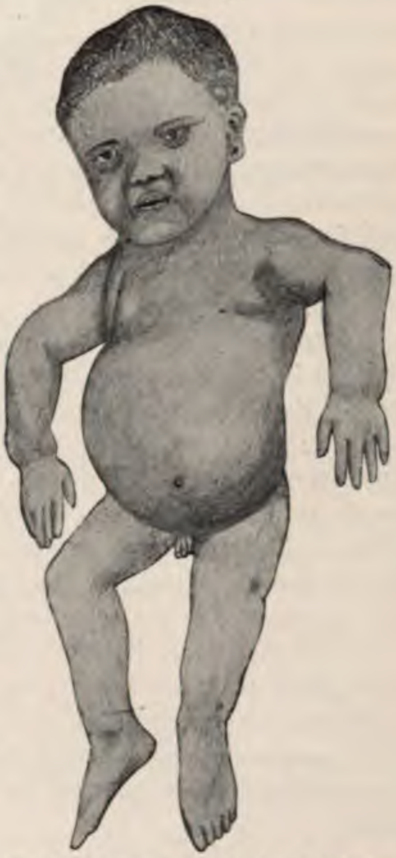
The globular head with enlargement, the head being disproportionately small, points to hydrocephalus.

A large head contracted from before backwards, with large face, thickened nose and lips, and puffy eyelids, is found in cretinism.

Hydrocephalus.—True hydrocephalus is dependent upon an accumulation of serum within the ventricles of the brain. It occurs in the majority of cases as a developmental condition, due to the closure of foramina connecting the ventricular cavities with each other, or to obstruction of the veins of Galen. It can only produce an enlargement of the head when it takes place in children whose fontanelles and sutures are still open. Under such circumstances the following appearances are to be noted :

The intracranial pressure leads to a pressing outwards of all the bones of the cranium, the bulging being especially noticeable in front. The entire cranium is enlarged and rounded, the face retaining its normal size. The fontanelles and sutures are widened. Sometimes the cranial bones are greatly thinned, and on palpation yield a crackling or parchment sensation. The appearance of the eyeballs is characteristic, these organs being forced outwards and downwards, so that the upper portion of the sclerotic is exposed. These local symptoms may or may not be preceded by or associated with other symptoms, including irritability, general weakness, paralysis, and even convulsions. Ultimately the mental condition becomes sadly deficient. Sometimes there is blindness from optic-nerve atrophy or neuritis. The increased size of the head necessarily adds to its weight, and if, as is usually the case, the muscles of the neck are weak, the head falls forwards, because the child is unable to maintain it in an erect position. It is not infrequent for the hydrocephalic head to have a circumference of from 24 to 30 inches, and Cruikshank has reported an extreme case in which it measured 52 inches.

FIG. 173.



Rachitis—showing shape of head, abdomen, and bones about the joints.

Rachitis.—The bone-changes ot rachitis are dependent upon an active proliferation of the cellular elements associated with abnormal calcification, these pathological processes being especially prominent in the long bones and in the bones of the skull. The growth of bone is derived from its superimposed periosteum. This growth is disturbed in rachitis, giving rise to increased thickness, which in the case of the cranial bones is apt to be unevenly distributed. After these preliminary remarks we are prepared to understand the abnormal shape of the rachitic head.

The edges of the cranial bones undergoing proliferation become thickened. At the same time there is delayed ossification, hence leading to delayed closure of the sutures and fontanelles. Grooves are thus formed readily perceived on palpation, and sometimes sufficiently marked to be visible on casual inspection. Bossy swellings are formed especially on the frontal and parietal bones, and if not promptly absorbed become organized. It is to them that the so-called square head of rachitis is due. By comparison with the prominences of the frontal and parietal bones, the vertex is flattened. All rachitic heads, however, do not conform to the type above described. Thus we may encounter a type called dolichocephalic, in which the skull is elongated fore and aft; another in which there is marked flattening in the occipital region; and still another, of which asymmetry is the important feature.

A condition known as cranio-tabes sometimes accompanies rachitis. It will be described shortly when treating of "Depressions." (*Vide* p. 649.)

Cretinism.—This is a congenital condition analogous to myxœdema, being dependent upon absence of the thyroid gland. The cretinoid head is characteristically brachycephalic in type, that is broadened from side to side, with flattening of the top and back. The soft tissues are thickened, the principal evidence of which is found in the condition of the lips, tongue, alæ nasi, and eyelids.

Syphilis.—The mechanism by which syphilis is capable of producing changes in the shape and size of the head is a varied one. In some instances the deforming lesion is distinctly syphilitic; in others it is nonsyphilitic (*e.g.*, hydrocephalus and rachitis), but is the result of syphilis. The last-mentioned class of cases is mostly the result of hereditary disease; the former may be either inherited or acquired. In neither case is it possible to decide as to the basic character of the lesion by its objective features. We are forced to make a diagnosis on the associated conditions, together with a knowledge that the lesion under examination is capable of being produced, directly or indirectly, by syphilis. Fortunately, data for a positive opinion are rarely wanting, for syphilis is characterized by a multiplicity of lesions. Exception may, of course, be made in those cases in which the malformation disintegrates and ulceration results, when the true nature of the trouble is made manifest by the objective appearances of the affected parts.

The syphilitic malformations include gummata (which lesions may undergo disintegration), nodes (in which the inflammatory process has not become organized into bony tissue), and exostoses (in which organization has taken place and tissue resembling bone has been formed). Periostitis of the cranium is attended by a swelling which may be readily differentiated from tumor formation by its history and appearance, diagnosis being made still more certain by the history and course of the case (pain, suppuration, etc.).

It must always be borne in mind that enlargements and swellings on bones may result from non-syphilitic causes, the true nature of which, other than those occurring from traumatism, is not well understood.

Microcephalus.—The recognition of microcephalus is based entirely upon the size of the head, this term being applied to cases in which the head measures less than 16 inches. Sight must not be lost of the fact that exceptionally very small heads exist in which the diminutiveness is the only pathological feature. A physiological microcephalus is never prominent. Pathologically, the condition may be attended by perfect symmetry, or the head may be flattened from side to side, or fore and aft. In many instances,—in the vast majority, in fact,—there is premature closure of sutures and fontanelles.

The mechanism of the production of microcephalus is a mooted point. Undoubtedly it is a developmental foundation. Current opinion holds that there is, primarily, backward development of the brain, and that the small cranial cavity is compensatory. A minority opinion contends that premature ossification of the sutures prevents the proper expansion of the skull and stunts the growth of the brain.

Microcephalus is associated with symptoms indicating deficient brain-power, as idiocy, paralysis, convulsions and contractures.

Asymmetry.—Asymmetry of the skull is, in the majority of instances, a congenital defect. It may be limited to either the cranial or the facial bones. In the former case it is rarely attended by a normal degree of mental vigor; at the same time, it must be remembered that some of the exceptions to this statement are somewhat remarkable. Hemiplegic seizures in infancy from different pathological lesions are sometimes followed by arrested development of the cranial bones over the affected portion of the brain, in which case the origin of the deformity is made manifest by the associated spastic hemiplegia, convulsions, etc. Some few cases of cranial asymmetry are the result of the malnutrition provoked by syphilis, the lesions themselves not being syphilitic.

Facial asymmetry occurring without changes in the overlying soft parts possesses the same significance as osseous asymmetry of the skull and other portions of the body. In conjunction with atrophy of the overlying muscular structures, it results from nerve injuries in growing subjects. The condition in these cases is one of arrested development, and not one of atrophy of bone.

Localized Swellings of Various Origin.—These include growths or prominences involving (a) the soft parts, and (b) the cranium and pericranium.

Swellings involving the soft parts include

i. Emphysematous and gaseous tumors, characterized by their soft and elastic feel, and resonance on percussion. Emphysema is due to fracture extending into the middle ear and involving the Eustachian tube, or lacerations of the soft parts of the nasal cavities. Gaseous tumors, generally called pneumatoceles, occur about the frontal sinuses and mastoid processes, the contained gas having escaped from these cavities. They are due to defects of the outer bony walls of these cavities.

ii. Cysts, which include (a) the congenital, (b) atheromatous or wens. The latter sometimes undergo air-infection and suppurate, the resulting destruction of tissue giving rise to foul odor. (c) Dermoid cysts.

iii. Cornu cutaneum, a cutaneous horn resulting from hypertrophy of the epidermis.

iv. Fatty tumors (rare).

v. Fibromata (rare).

vi. Chondromata (very rare).

vii. Malignant disease, *e.g.*, carcinomata, sarcomata, etc., presenting the characteristics of this variety of tumor.

viii. Vascular tumors.

Swellings originating in the bony structures include

i. Hyperostosis, *i.e.*, a diffuse swelling of the cranial bones, consisting of compact bony tissue ; when involving the entire cranium, not diagnosable ; at times dependent upon syphilis or rachitis, but not infrequently occurring independently of these constitutional states.

ii. Osteomata, including circumscribed bony swellings from a variety of causes, including syphilis, rachitis, traumatism, etc. Cephalhæmatoma, due to subperiosteal hæmorrhage from obstetric injury, when the effused blood is not promptly absorbed, take on bony formations. The tumors thus formed rarely give rise to much disfiguration.

The Fontanelles and Sutures in Infancy and Childhood.—The principal sutures of the skull are ossified anywhere from the sixth to the tenth month. The posterior fontanelle is usually closed by the end of the second month ; the anterior fontanelle remains open until the fourteenth to the twenty-second month. The close of sutures and fontanelles may be delayed by reason of rickets, congenital syphilis, and cretinism. In the latter condition, the fontanelles have been known to remain open until adult life.

Bulging of the fontanelles is symptomatic in cerebral hæmorrhage of the new-born, hydrocephalus, and tubercular meningitis.

Separation of the sutures is observed in premature and syphilitic infants, and in cases of hydrocephalus.

Depressions.—The error of concluding that a fracture of the cranial bones exists when a depression is found after traumatism is altogether too prevalent. It is well to bear in mind that an adherent cicatrix may produce a depression which is capable of deceiving the most skillful. Then, again, the absence of a depression cannot be taken as evidence that a fracture has not taken place, as instanced by the many such accidents discovered by exploratory incision in the Hahemann Hospital.

Depressions from maldevelopment of the cranial bones are rare (*aplasia cranii*). Sometimes the opening in the skull leads to the formation of a tumor (*meningocele*). The parents of these patients are usually poorly nourished, and sometimes syphilitic.

Depressions may result from disappearance of the cranial bones at one or more spots (*atrophia seu anostosis cranii*). It seems to occur with greater frequency in the aged, and is a part of a senile atrophy which may invade other portions of the body.

Separation of cranial sutures presents a depression along the line of a suture. It is due to violence, especially in young subjects, and is generally associated with fracture, particularly at the base of the skull.

Syphilitic defects of the external table are for the most part the result of deep gummata.

Rachitic cranio-tabes is a condition dependent upon rachitis, and exhibits one or more defects in cranial ossification, the defect being closed in by fibrous tissue.

The Face.

It is a difficult matter, in a work like the present, to describe the variations in the appearance of the face, and their diagnostic relations. The knowledge is of such a character that it can be best acquired by observation at the bedside. The study of the patient's facial expression will ofttimes open a flood of light on a hitherto dark problem. Thus, a peculiarly placid expression, entirely devoid of any evidence of suffering or malnutrition, will be presented, while the patient herself uses the most graphic language possible in depicting her sufferings as beyond all description ; and immediately we feel assured that our patient has hysteria, unless we can account for her symptoms in some other way.

The expression of the face in painful affections gives us a guide as to the severity of the suffering. But we must not be led too much by this, for the personal equation as to the ability to bear pain is a most important factor, for some people endure agonizing pain with fortitude, while others cannot tolerate the prick of a pin without fainting.

An apathetic expression or one of relaxation is observed in some cases of typhoid. It is to be regarded as evidence of the lack of muscular tone.

An expression of anxiety attends paroxysms of dyspnœa and angina pectoris.

A peculiar expression of fixity or stolidity is characteristic of paralysis agitans. One who has seen a number of cases of this disease is frequently enabled to diagnose the case by the face alone.

In dyspnœa from any cause the patient holds his mouth wide open, the alæ nasi moving regularly with each respiration. The deficient aëration of the blood causes the face to assume a bluish or dirty hue.

The Hippocratic face is the face of impending death. It is especially observed, with all its characteristic features, in cholera Asiatica and acute peritonitis. To use the words of the great master of medicine—"sharp nose, hollow eyes, collapsed temples; the ears cold, contracted, and their lobes turned out; the skin about the forehead being rough, distended and parched; the color of the whole face being brown, black, livid or lead-colored."

Reference has already been made to the diagnostic significance of œdema of the eyelids. Œdema of any portion of the face, but especially about the eyes, may be occasioned by urticaria or angio-neurotic œdema.

Localized œdemas may be due to inflammatory changes of the parts beneath, as follows:

Over the upper jaw, to alveolar abscess, phosphorus necrosis, or disease of the antrum or maxillary sinus.

Over the lower jaw, alveolar abscess or actinomycosis.

Over the mastoid, to mastoiditis or thrombosis of the lateral sinus.

Over the forehead, in glanders, or thrombosis of the superior longitudinal sinus.

Movements of the Face.—The movements of the face may be impaired by reason of loss of power (paralysis), or by excessive action (spasm). Paralysis of the face may result from disease of the facial fibres above the facial muscles (supranuclear palsy), from disease of the nucleus itself, or from lesion of the nerve-fibres after leaving the nucleus (peripheral).

Supranuclear or cerebral facial palsy presents important features by which it may be readily differentiated from the nulear and infranuclear varieties. In the first place, the paralysis of the face is associated with paralysis of the corresponding arm or leg, and there are symptoms associated to show that the functional integrity of the cerebrum is disturbed. The upper facial muscles, *i.e.*, the occipito-frontalis, the orbicularis palpebrarum, and the corrugator supercilii, escape. The paralysis seems to be less marked during emotional movements of the face than during voluntary movements.

Paralysis of the facial muscles from disease of the nucleus is of rare occurrence. It is then associated with paralysis of muscles tributary to the adjacent nuclear cells.

Peripheral Facial Palsy; Paralysis of the Seventh Cranial Nerve; Bell's Paralysis.—Disease of the facial nerve (neuritis) may result from disease of the middle ear or from exposure to cold, the latter being the more common cause. Some few cases result from traumatism, as from forceps-pressure during delivery, and incisions during surgical operations. Syphilis is only exceptionally a cause of uncomplicated facial paralysis.

In the majority of cases of facial paralysis the onset is rapid. Within a comparatively few hours the patient loses all control over the muscles of the face. The paralyzed side is smooth, flaccid and expressionless. Because of the involvement of the occipito-frontalis and corrugator supercilii, all furrows and wrinkles are obliterated from the brow. The orbicularis palbebrarum is paralyzed, and so the eye stands wide open, even during sleep. The conjunctiva, no longer protected from dust and foreign particles floating in the air, may become inflamed and painful. The tissues of the face, hanging loose, draw down the lower lid, producing ectropion. The buccinator is paralyzed ; hence, during the act of chewing, food tends to escape from between the teeth and lodge between the gums and the cheek. The mouth is drawn towards the healthy side, while the angle of the paralyzed side is depressed. The behavior of the affected muscles under the stimulus of galvanism and faradism is of great importance. The reaction of degeneration is a most valuable means for demonstrating that the lesion is nuclear or infranuclear.

Paralysis of the Muscles of Mastication; Fifth-Nerve Palsy.—Paralysis of the motor branch of the fifth nerve may be due to disease within the pons (generally some focal lesion other than degeneration), certain diseases at the base of the brain, as tumors, meningitis, etc., and disease in the course of the nerve itself. Of the latter class of lesions we have neuritis and traumatism. The former almost always occurs as secondary to other morbid processes, although occasionally it results from exposure to cold, syphilis, or gout. The form of neuritis which is associated with or lies at the bottom of herpes zoster is very common in this nerve. Traumatic lesions of the trigeminus are rare as complicating fracture of the skull or direct blows, but occur occasionally as the result of gunshot wounds through the mouth or nasal cavity. These same causes, affecting the sensory fibres of the nerve, produce anæsthesia.

Paralysis of the muscles of mastication requires a little care for its detection when the loss of power is slight. The patient should be directed to close his jaws firmly. The examiner at the same time places his fingers over the contracting muscles on both sides, and notices their relative powers. In mild cases he may note only that the muscles on the diseased side contract somewhat more slowly than those on the other. The temporal, masseter and pterygoid muscles are paralyzed. That of the former two is shown by deficient power of mastication on the affected side ; that of the pterygoids by inability to move the lower jaw towards the healthy side. Masticatory paralysis is very frequently bilateral. When due to disease of the nerve itself, the characteristic reaction of degeneration is present. Subsequently the muscles waste.

Facial hemiatrophy is an affection of unknown etiology characterized by wasting of one lateral half of the face, in which the atrophy involves particularly the bones and the connective tissues. The overlying tissues cover these tightly, and so fit into all depressions. Thus, the affected side presents an expression as much different from the other as if it belonged to a different individual. The muscles of the face are neither paralyzed nor atrophied. Sensory disturbances appearing in the shape of anæsthesia may be present in any or all parts supplied by the fifth nerve. The skin is very much wasted, to the extent even of being one-tenth of the thickness of the normal. Facial hemiatrophy is to be differentiated from atrophic palsies limited to the muscles supplied by the fifth and seventh nerves and congenital facial asymmetry. In the former, the paralysis with the reaction of degeneration and the predominance of muscular over osseous atrophy furnish all-sufficient distinctions. In congenital asymmetry, the difference is never so great as in facial hemiatrophy.

The prognosis of facial hemiatrophy is absolutely unfavorable as regards possibility of cure.

Abdominal Enlargements.

Enlargements of the abdomen may be either *general* or *circumscribed.* Of the general enlargements of the abdomen, those arising from flatulent distention and abnormal accumulation of adipose have been mentioned elsewhere, leaving only for consideration at this time abdominal dropsy or ascites.

Ascites.—The symptoms and physical signs of ascites are consistent with what one would expect from the distention of a cavity with yielding walls by fluid. If the accumulation is not sufficiently great to completely distend the abdominal cavity, the shape of the abdomen will vary according to the position of the patient. When lying down, the lateral portions of the abdominal walls will bulge as the result of the weight of the contained fluid. If, on the other hand, the quantity of effusion is at a maximum, the anterior surface of the abdomen will be prominent or dome-like, and will retain this shape even though the patient assume the erect posture. Palpation gives a sense of semi-fluctuating resistance, which to the experienced hand is unlike any other sensation observed in cases of abdominal enlargement from causes other than cystic growths.

The percussion signs are altered with the position of the patient. When he is lying on his back, there will be dullness over the flanks, while the anterior abdominal surface will be tympanitic. When the patient changes his position, the lines of dullness are found over the most dependent portions of the abdominal walls. In doubtful cases the patient may be placed in the knee-chest position, when dullness will be elicited over the umbilicus, where, when he was lying upon his back, tympanitic resonance existed.

In the case of women, an early diagnostic sign may be obtained by vaginal examination. With a small amount of effusion and the patient in the lithotomy position, the uterus will appear to palpation to be resting upon a water-bed. With the pelvis elevated, this sign disappears.

To detect ascitic accumulation by palpation of the abdomen, it is a well-known procedure to have an assistant press the border of one of his hands upon the patient's linea alba. This prevents the transmission of any vibrations of the abdominal walls. The observer then places one hand over the effusion on one side, while he taps at the corresponding point on the opposite side with the fingers of his free hand. If fluid is present, a distinct wave will be transmitted to the palpating hand.

Ascites may result from a variety of pathological lesions, the differential diagnosis of which is well stated in the following quotation from Bury:

"I. Ascites **not associated with dropsy** elsewhere:

"1. Abdominal pain and tenderness are severe; the abdomen is usually much enlarged from distention of the intestine, but the quantity of ascitic fluid is usually small, and insufficient to give distinct fluctuation. Vomiting, constipation, pyrexia and collapse are other marked features of the case.

"*Acute Peritonitis.*

"2. Abdominal pain and tenderness are moderate or slight.

"(a) Tumors of various sizes, shapes and consistence may be felt.

"*a*. The abdomen is retracted or moderately enlarged. The patient is usually young, is emaciated, subject to night-sweats, and to irregular action of the bowels. There may be signs of tubercle in the lungs, or in other organs. Frequently there is redness and œdema about the umbilicus.

"*Tubercular Peritonitis.*

"*β*. The abdomen is greatly enlarged; there is vomiting and emaciation, and the patient is usually past middle life.

"(1) Fluctuation is distinct, and nodules may be felt near the umbilicus; there may be evidence of cancer of the stomach, ovary, or other organ. *Cancer of Peritoneum.*

"(2) Fluctuation is indistinct; hard, irregular, or gelatinous masses may be felt; the fluid obtained by tapping is viscid or thin and turbid, with blood and colloid cells.

" *Colloid Disease of Peritoneum.*

"(b) No tumors to be felt; the abdomen is moderately enlarged, and constitutional disturbances are usually moderate in degree. *Chronic Peritonitis.*

"3. Abdominal pain and tenderness slight, absent, or limited to hepatic region.

"(a) There is much ascites; the superficial veins are distended; there are venous stigmata on the cheeks; the liver is sometimes palpably enlarged, sometimes not, and dullness may be diminished; the spleen is often enlarged. *Cirrhosis of the Liver.*

"(b) Degree of ascites variable; superficial veins not usually enlarged; jaundice commonly present; irregular or nodular enlargement of liver; spleen not enlarged.

" *Cancer of Liver.*

"II. Ascites **associated with dropsy** elsewhere:

"1. Dyspnœa and œdema of legs before the occurrence of and out of proportion to the degree of ascites.

"(1) Physical signs of dilatation of right ventricle. *Tricuspid incompetence from mitral disease, or from bronchitis and emphysema, or from weakness of cardiac muscle.*

"(2) Rapidly progressing œdema, great enlargement of superficial veins of chest and abdomen, and perhaps physical signs of a tumor or indications of pressure upon œsophagus or lungs.

" *Tumor Compressing Inferior Vena Cava.*

"Œdema beginning in the face, and often associated with hydrothorax, albuminuria, and other signs of renal disease.

" *Kidney Disease.*"

The character of the fluid in ascites varies according to its cause. In ordinary dropsy, *i.e.*, non-inflammatory, it is a clear, yellowish fluid, having a specific gravity of about 1015, and containing not more than 2.5 per cent. of albumin. When the effusion is inflammatory, the specific gravity is somewhat higher than above stated (1018), and the percentage of albumin amounts to 4.5. The admixture of blood with the fluid is strongly suggestive of malignant disease of the peritoneum. Too much importance must not be attached to this symptom, as it is not invariably present in malignancy, and it may occur in conjunction with other lesions.

Chylous ascites is characterized by the presence of a milky fluid, the opacity being due to the presence of fat. It may result from perforation of the thoracic duct, filiarasis, carcinoma, and (as Osler has suggested) an exclusive milk diet.

Circumscribed abdominal enlargements may be due to increase in size of the abdominal viscera, as the liver, spleen and kidneys, or to misplacements of the same, or to new formations. A special variety of tumor, designated by the term "phantom," is not so very uncommon. As a rule, such prominences are dependent upon muscular contractions. In many cases they are of purely hysterical origin. In still others they are the result of irritation of one of the abdominal viscera, giving rise to reflex muscular contraction or local intestinal pneumatosis which disappears with the primary disease.

There are very few problems in diagnosis more difficult than the determination of the nature of an abdominal growth. Empirical methods, or the matching of symptoms given in the books with those of the case in hand, will not bring success. An accurate conclusion is only possible after a thorough examination of the case, and the careful weighing of all signs and symptoms. In following this plan the reader will find considerable assistance from the following quotations from Leftwitch's Index :

Central Tumors :

Aneurysm ; cirrhosis of the stomach ; malignant disease of the stomach, pancreas, omentum or intestines ; fatty tumor of the omentum ; hypertrophy of the pylorus ; intussusception ; enlargement of the lumbar glands ; enlargement of the mesenteric glands ; mesenteric cysts ; thickened peritoneum ; retroperitoneal abscess ; pancreatitis.

Tumors Traced into the Pelvis :

Congestion of uterus (chronic) ; cyst of broad ligament ; distended bladder ; ectopic gestation ; fibroid tumor of the uterus ; fibrocystic tumor of the uterus ; hydrometra ; hydrosalpinx ; hypertrophied bladder ; hæmatosalpinx ; myoma ; retained menses ; ovarian cyst ; pregnancy ; pyosalpinx ; polypus ; peritoneal hydatids ; subinvolution ; tubal cancer or tubercle.

Wandering Tumors :

Concretions in the intestines ; cancer of the pylorus ; fatty tumor of the intestines ; floating kidney ; floating spleen ; floating lobe of liver ; impacted fæces ; phantom tumor ; tumor of transverse colon.

Lateral Tumors :

Appendicitis ; cystic kidney ; dysentery (doughy colon) ; encephaloid kidney ; fæcal accumulation ; distended gall-bladder ; hydronephrosis ; hydatids of liver of kidney ; pelvic hæmatocele ; hydrosalpinx ; hæmatosalpinx ; ovarian cyst ; pelvic abscess ; perinephritic abscesses ; pyonephrosis ; parasite of the kidney ; enlarged spleen ; sarcoma of kidney.

Fluctuating Tumors:

Ascites; abscess of abdominal walls; distended bladder; ectopic gestation; effusion into lesser peritoneal cavity; hydatids; hydronephrosis; hydrosalpinx; lumbar abscess; ovarian cyst; pregnancy; pyosalpinx; pyonephrosis.

Enlarged Mesenteric or Lumbar Glands:

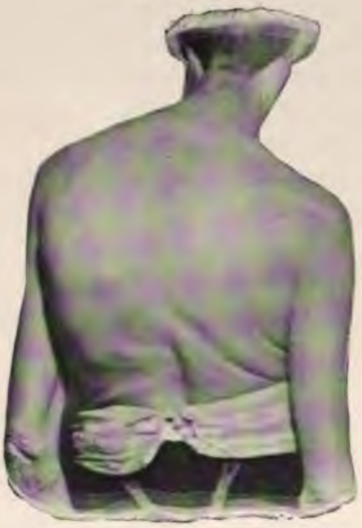
Amyloid disease; cancer of intestine, kidney, testis, etc.; dysentery; enteric fever; glandular fever; peritonitis; tuberculosis.

Alterations in the Shape of the Back.

The principal alterations in the shape of the back include

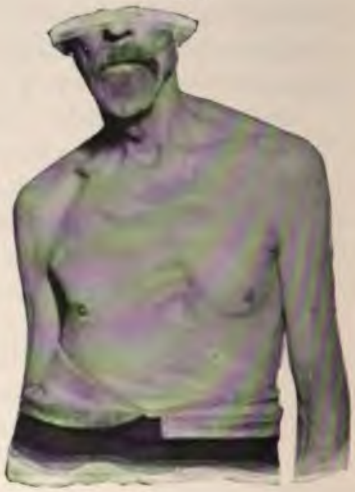
a. **Kyphosis,** which is a posterior curvature of the spine, and may be due to

FIG. 174.



Aggravated lateral curvature of spine in shoemaker, with deformity of chest.

FIG. 175.



Anterior view of same case as Fig. 174.

- i. Caries of the vertebræ, the deformity being angular.
- ii. The stooping of age.
- iii. As part of the changes of the emphysematous chest.
- iv. Mollities ossium.
- v. Arthritis deformans.

b. **Scoliosis,** the curvature being rotary-lateral, and which may be due to

- i. Faulty position among school-girls.
- ii. Unequal length of the lower extremities.
- iii. Rachitis.
- iv. Cerebral and spinal paralyses.
- v. Intrathoracic disease.

c. **Lordosis,** in which the normal convex curve of the spine is exaggerated, and may be due to
   - i. Pregnancy, ascites and abdominal tumors, being produced by the abdominal weight in these conditions.
   - ii. Pseudo-hypertrophic paralysis.

Alterations in the Shape of the Extremities.

Alterations involving the extremities may be studied as involving the (a) soft parts, including muscular tissue, adipose, connective tissue, and skin; (b) the bones; (c) the joints.

Studying first the muscular changes, we find that the muscles may be

I. Enlarged (true or false hypertrophy).
II. Diminished in size (atrophy of disuse, or numerical atrophy).

1. **Enlargement of the Muscles.**

i. PSEUDO-HYPERTROPHY, observed in a form of muscular dystrophy known as pseudo-hypertrophic paralysis; a disease of developmental origin; starting in early childhood; apparent hypertrophy of certain muscles, *e.g.*, those of the calves and shoulders, associated with atrophy of others; exaggeration of the dorso-lumbar concavity; patient exhibits a waddling gait; when rising from lying on the floor, goes through the process known as "climbing up the thighs;" sensory disturbances absent; course very chronic; incurable; death occurs from intercurrent disease.

ii. TRUE HYPERTROPHY is rare as a pathological condition. Occurs in Thomsen's disease (myotonia congenita),—a hereditary defect characterized by spasm or rigidity of muscles as soon as they are put into action, relaxation taking place in a few seconds.

iii. INFLAMMATORY SWELLING OF THE MUSCLES. Common as a secondary phenomenon to traumatism, and to inflammation in adjacent structures. Polymyositis may occur as a primary affection. Characterized by swelling of the affected extremities, so that they lose their normal contour, the outlines of individual muscles being obliterated. On palpation, different consistence in different parts, according to grade of inflammation; sometimes doughy, sometimes hard and distended. May be associated with cutaneous œdema. Skin may take on increased color, which has been described as erysipelatous. Spontaneous pain severe, greatly aggravated on manipulation. Tendency to involve the trunk-muscles before those of the limbs, and of the proximal before the distal muscles of the extremities. Prognosis very unfavorable, death resulting from involvement of muscles of respiration.

Muscular atrophy may proceed from

a. Disuse of the parts.
b. Disease of the upper motor neuron.
c. Disease of the lower motor neuron.
d. Disease of the muscles themselves.
e. Disease of joints.

The **atrophy of disuse** is rarely extreme, and is due entirely to disturbance of nutrition. It disappears as soon as the affected part is made active. Some of the cases of muscular atrophy attendant upon joint-disease belong to this class.

The extent of the atrophy associated with lesions of the **upper motor neuron** varies greatly, according to circumstances. In the majority of cases the muscular wasting is no greater than is to be expected from cessation of function. Moreover, it takes place only after the lapse of considerable time. In the case of infants and children, the atrophy proceeding from cerebral lesions and attendant upon paralysis is often extreme. In most instances it is in great part dependent upon arrested development, all the structures of the affected member being much smaller in every measurement than its fellow of the opposite side. (*Vide* Spastic Hemiplegias and Diplegias of Infancy and Childhood.)

It is in diseases of the **lower motor neuron** that the most remarkable examples of muscular atrophy are found. There is a growing tendency to place the atrophies apparently dependent upon idiopathic muscular degeneration in this class, it being claimed that improved histological technique shows disease of the lower neuron. The affections universally conceded as belonging to the class under study are :

i. Acute poliomyelitis anterior.
ii. Chronic poliomyelitis anterior (spinal progressive muscular atrophy), and its congeners, progressive bulbar paralysis, nuclear ophthalmoplegia, and amyotrophic lateral sclerosis.
iii. Syringomyelia.
iv. Multiple neuritis, proceeding usually from toxic causes.
    i. As a sequela to or accompaniment of certain diseases, *e.g.*, diphtheria, typhoid fever, or diabetes, or the result of
    ii. Poisons introduced from without, as alcohol, lead, mercury and arsenic.
v. Arthritic muscular atrophies (other than those above stated).

i. **Acute Poliomyelitis Anterior.**—This disease in the majority of cases attacks children ; adults but occasionally. It seems to be especially frequent during hot weather. There is a growing sentiment in favor of its infectious origin. Its onset is characterized by constitutional symptoms, which, however, are not diagnostic. They include fever, general convulsions, and delirium. Then comes the paralysis, attended by flaccidity of the affected muscles. The legs or all four extremities may be involved. Nearly always, one limb is more severely affected than the others; or the brunt of the disease may be borne by single muscles or groups of muscles. Within a few days (a week or thereabouts) atrophy begins, and often proceeds to an extreme degree. There is no disturbance of sensation or involvement of the sphincters. Both deep and superficial reflexes are abolished. In the course of a couple of weeks, unless the case be a severe one, the paralysis disappears in part. Some muscles are nearly always permanently affected. Deformities in the shape of club-foot are apt to remain. The pathological lesion is an inflammation of the ganglion cells of the anterior gray cornua.

The clinical picture of acute poliomyelitis is so well defined that diagnostic difficulties would seem to be impossible. Yet such are not infrequent. It has been confounded with *rachitic pseudo-paraplegia*. In the latter condition there are the characteristic rachitic symptoms,—bending of the ribs, enlarged and painful joints, and hyperæsthesia of the extremities; movements are possible, but painful; there is no muscular atrophy, and the electrical reactions are normal, as are the deep and superficial reflexes. Diffuse sweats are commonly present.

Spastic spinal paralysis lacks the reaction of degeneration, the muscular atrophy, and the relaxed condition of the paralyzed muscles. It is of slow onset. Deep and superficial reflexes are greatly exaggerated. All of the paralyzed parts exhibit a spastic condition.

From *multiple neuritis* acute poliomyelitis is differentiated by the mode of onset, which in neuritis is rarely as rapid as in spinal affection. The muscles paralyzed show a relation to certain nerve-trunks, and are not grouped according to the representation of centres in the spinal cord. Sensory functions are impaired whenever a mixed nerve is involved.

ii. **The spinal form of progressive muscular atrophy** is dependent upon degeneration of the ganglion cells of the anterior gray cornua. If the pathological process invades first the ganglion cells, *i.e.*, the lower neuron, the initial symptoms are muscular atrophy with corresponding loss of power in the affected parts. The upper extremities are nearly always the first attacked, the atrophy commencing in the thenar and hypothenar eminences. Still, this is by no means necessary, for the shoulder muscles may be the first involved. Beginning at any point it may, the pathological process extends until eventually the entire body may become involved. The patient consists truly of but skin and bones. Sensibility is unimpaired. The superficial and deep reflexes of the lower extremities are normal or increased. The sphincters are not usually affected.

If, however, the pathological process begins in the upper neuron, *i.e.*, in the pyramidal tracts, paralysis with rigidity marks the onset of the disease. With involvement of the anterior gray cornua, muscular atrophy is noted. To this condition the special designation amyotrophic lateral sclerosis has been applied.

This **amyotrophic lateral sclerosis** is by many regarded as simply one of the varieties in the clinical course of spinal muscular atrophy. Nevertheless, as it presents itself as a distinct clinical type, and seems to run a more rapid course than progressive muscular atrophy, a retention of the term is convenient. The prominent symptoms, spasticity and muscular atrophy, vary in prominence according as the brunt of the disease is borne by the pyramidal tracts or anterior cornua respectively. The first symptoms include formication and numbness of the fingers, followed by motor weakness in association with muscular atrophy. Next comes the muscular rigidity, which may attain such a degree of severity as to amount to absolute contracture. In the second stage of the disease the legs become involved, paraplegia with rigidity being the prominent symptom. In the terminal stage, the patient presents the characteristic phenomena of bulbar palsy. Throughout the course of the disease the deep reflexes are increased. The knee-jerks are exaggerated, and the tendon reflexes are readily obtainable at the elbows and wrists. Ankle clonus is present. In some cases the spastic phenomena are absent, in which case the same increase in the tendon reactions is observed. The spasticity tends to diminish as the muscular atrophy increases.

Still a third mode of onset is that in which the degenerative changes start in the nerve nuclei in the medulla, as a result of which the initial symptoms constitute paralysis of the muscles of the lips, tongue, pharynx, and larynx (progressive bulbar palsy, progressive glosso-labial paralysis). Later appears the general muscular atrophy, which, if life is spared long enough, proceeds to a complete loss of muscular substance.

Progressive Bulbar Paralysis; Glosso-Labio-Laryngeal Paralysis.—Reference has several times been made to this affection when speaking of symptoms of the mouth, throat and larynx. It now remains to present the complete clinical picture of the disease. The onset of the disease is always slow, the first manifestations scarcely, if ever, being of a character to denote the serious group of phenomena which are about to follow. These include such symptoms as pain in the nape of the neck, vertigo, general languor, etc. The loss of power over the lips and tongue is first indicated by a certain difficulty in talking or by vocal fatigue after speaking for more than an ordinary length of time. Difficulties in articulation assert themselves, varying according to the muscles first attacked. Usually the tongue and the lips are the first to suffer. Finally dysphagia appears. Then there may be especial difficulty in swallowing liquids or solids, or both, according to circumstances. If the tongue is seriously disabled, the patient will find it impossible to properly prepare the bolus of food, or even to push it back into the throat, without the aid of the hands. If the muscles of the glottis are involved, attempts at swallowing liquids will be accompanied by choking and strangling. The attempt at swallowing liquids may be succeeded by regurgitation of the same through the nose. When the laryngeal muscles are invaded, the voice becomes impaired, and even lost. Saliva cannot be retained in the mouth. All of the paralyzed muscles undergo atrophy, and the local reflex excitability of the parts is more or less destroyed. Common sensibility remains unimpaired. The terminal stage of the disease is not uncommonly attended by extensive atrophy of the muscles of the extremity.

It is very exceptionally that ophthalmoplegias mark the onset of a general muscular atrophy.

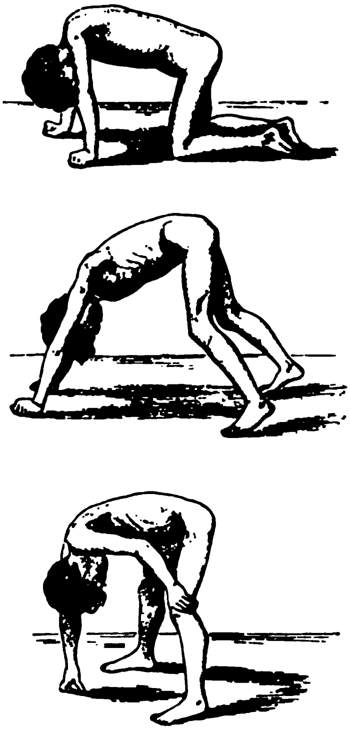
The Muscular Dystrophies.—Under this generic term were included, for a number of years, several types of muscular atrophy believed to be essentially myopathic in origin, and exhibiting as common characteristics appearance in childhood, a tendency to attack several members of the same family, and by the absence of fibrillary twitchings and the reaction of degeneration. Improved technique in neuro-pathology has demonstrated that all of these conditions are really attended by changes in the lower motor neuron; hence their myopathic origin can no longer be entertained. The affections included in this class include the following:

a. Pseudo-hypertrophic paralysis.
b. Erb's juvenile muscular atrophy.
c. Ladouzy-Dejerine type of muscular atrophy.
d. Peroneal type of Charcot, and Marie and Tooth.

(*a*) **Pseudo-Hypertrophic Paralysis.**—This was the first described of the so-called myopathies. Its first symptoms appear at about the age of four years, and never after the twentieth year of age. As a rule, the first sign of any difficulty is observed when the child begins to walk, when attention is called to certain peculiarities in its gait. At first there is a tendency to fall from trifling causes. Next a peculiar waddling gait is noticed. When standing, the child is observed to tilt the pelvis unduly forward, causing the abdomen to become abnormally prominent and the dorso-lumbar concavity of the spinal column exaggerated. The upper portion of the body is thus thrown so far backwards that a plumb-line let fall from between the scapulæ clears the sacrum by two three inches. When the patient endeavors to rise after sitting on the floor, he does so, as already stated, by the process known as "*climbing up the thighs*" (Fig. 176). Certain muscles, notably those of the calves, present undue prominence, giving an idea of strength. Other muscles, however, exhibit marked atrophy. The sphincters are never involved. Sensory disturbances are absent. The knee-jerks and electrical reactions are normal in the early stages of the disease. The prognosis is absolutely unfavorable.

(*b*) **Erb's Juvenile Muscular Atrophy.**—The majority of cases of this type also appear in early life, though, exceptionally, its first manifestations are delayed until the patient is pretty well advanced in years. The muscles about the shoulder are first affected. Later, those of the upper arm become involved, including especially the pectorals, trapezii, rhomboids, serrati, latissimus and longissimus dorsi. The muscles of the forearms, the deltoids, and supra- and infraspinati usually escape. When the atrophy finally invades the lower extremities, the muscles about the hips and thighs are first affected. The legs do not suffer until late, and may even escape entirely. The peculiarities of gait and posture observed in pseudo-hypertrophic paralysis are frequently present.

FIG. 176.



Climbing up the thighs in pseudo-hypertrophic paralysis.—After Gowers.

(*c*) **The Landouzy-Dejerine Type of Muscular Atrophy.**—Owing to the distribution of the muscular atrophy, this has also been known as the *facio-scapulo-humeral type* of muscular atrophy. It usually begins in childhood at an earlier age than Erb's muscular atrophy, but, like it, may develop later in life. The atrophic process begins first in the face. The wasting of the orbicularis oris causes the lips to protrude (*tapir mouth*). The muscles of the eyes, deglutition and mastication escape. The atrophy extends to the muscles of the shoulders and arms, and pursues the course of Erb's type of muscular atrophy.

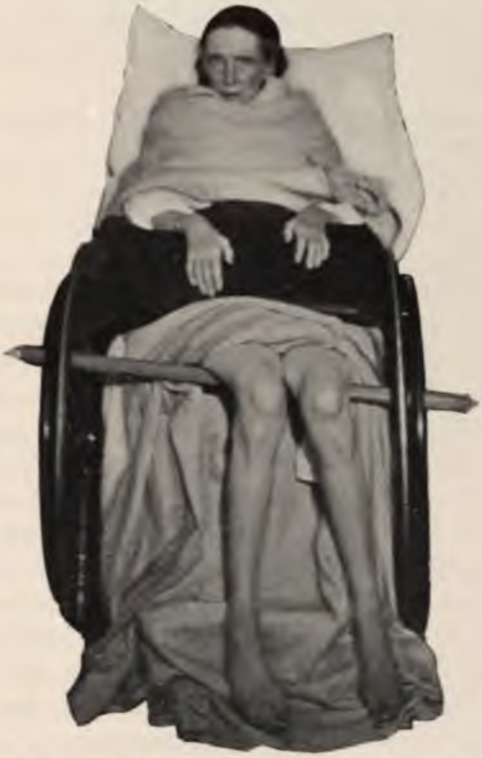
(*d*) **Peroneal Type of Charcot and Marie and Tooth.**—In this variety, the atrophy begins first in the extensor digitorum communis, the extensor longus pollicis, or the peronei muscles, whence it gradually extends. Sometimes the intrinsic muscles of the foot are affected ; in still others, the gastrocnemius. The unequal wasting of the muscles gives rise to various forms of club-foot. The disease spreads symmetrically, involving the upper extremities after many years.

Unlike the other varieties of so-called muscular dystrophy, fibrillary twitchings and a modified reaction of degeneration are noted. Sensibility is occasionally impaired, and pain is present. The reflexes are altered according to the distribution of the atrophic process.

iii. **Syringomyelia.**—Muscular atrophy is not the important clinical feature of syringomyelia; still it is present to some extent in practically all cases. Its location will depend entirely upon the portion of the spinal cord involved. Usually it is in one of the upper extremities. When bilateral it is by no means symmetrical. It is associated with the symptom on which the diagnosis of syringomyelia is based, namely, the dissociation of cutaneous sensations.

iv. **Multiple Neuritis.**—In this disease muscular atrophy is present, but its recognition depends upon the grouping of the paralyses, the presence of anæsthesia when sensory nerves are involved, and the presence of one of the well-known causes of neuritis. Muscular atrophy does not take place until the paralysis has become an obtrusive phenomenon. (*Vide* Paralysis of Multiple Neuritis.)

FIG. 177.



Extreme muscular atrophy, with "dropped foot" and "dropped wrists" from multiple neuritis, due to puerperal infection.

v. **Arthritic Muscular Atrophy.**—Muscular atrophy may be brought about in various ways as a result of joint-disease. It is understood that the enforced rest of the inflamed limb results in simple atrophy of the muscles. Again, the joint-inflammation may involve the contiguous structures, including the nerves. The neuritis thus engendered produces its characteristic muscular atrophy with the typical reaction of degeneration.

Joint-Lesions.—The diseases which may give rise to joint-enlargements include the following:

a. Acute inflammatory rheumatism.
b. Chronic articular rheumatism.
c. Gout.
d. Arthritis deformans.
e. Gonorrhœal arthritis.
f. Scarlatinal synovitis.
g. Simple synovitis.
h. Hæmophilia.
i. Syphilis.
j. Tuberculosis.
k. Spinal-cord disease.
l. Rachitis.

(*a*) **Acute Inflammatory Rheumatism; Rheumatic Fever.**—Acute inflammatory rheumatism is ushered in by fever, associated with pains in the joints and extremities. In some cases the onset is preceded by a period of general ill-health of indefinite character, and sometimes by pharyngeal or tonsillar inflammation. The fever and pains gradually increase in severity, so that at the end of two or three days the symptoms of the disease are well-developed. The fever, as a rule, is moderate, rarely rising above 103° F., excepting in cases of complications or rheumatic hyperpyrexia. The articular changes usually appear in one of the larger joints, notably in the knees, ankles, hip, shoulders, elbows, and wrists. It is the usual course for one or more joints to bear the brunt of the disease at one time. After two or three days the swelling and inflammation disappear, and other joints become involved. And so the lesions alternate among the various joints until recovery takes place.

The affected joints are red and swollen, and on palpation exhibit local increase of temperature. The parts are excessively painful, especially to the slightest manipulation. The affected parts are kept rigid, and in an attitude that will give the least discomfort. Thus, it will be observed that the knees and elbow are held flexed, while the wrists and ankles are extended.

Profuse sweating is a common attendant upon rheumatic fever. It is usually described as possessed of a sour odor, which by some is regarded as diagnostic. While there can be no doubt of the frequency with which sweating of a sour odor is present, the diagnostic value of the symptom is to be questioned. Current views concerning the origin of rheumatism, attributing the disease to cold, lead attendants to wrap patients in flannels, and avoid the use of cleansing baths. Under such circumstances, it is not surprising that the sweat should become offensive. In hospital cases who are bathed regularly, this sour odor is not observed. Sudamina are frequently present.

Inflammatory rheumatism is noted for the number and importance of its complications. Especially frequent are acute inflammatory lesions of the heart, as endocarditis, pericarditis, and myocarditis. Indeed, the physician who carries a case through with a sound heart has every reason for self-congratulation.

Cases treated on the expectant plan usually run a course of six to eight weeks. Efficient treatment in the majority of cases reduces this period two weeks.

The diagnosis of rheumatic fever should present no difficulties, excepting when that disease occurs in childhood. Childish rheumatism, it must be remembered, presents several features peculiarly its own. In the adult, rheumatism involves characteristically the joints. In children, the joints are but slightly affected ; indeed, they frequently escape. The abarticular manifestations of rheumatism are especially prominent in children. Thus, we find endo- and pericarditis an almost invariable complication. Indeed, these may appear without any joint-lesion. Subcutaneous nodules are far more frequently observed in children than in adults. They are commonly associated with cardiac inflammations. They generally appear about the tendons, and vary in size from that of a hemp-seed to that of an almond. They are not apt to be tender, nor is there any reddening of the overlying skin excepting from accidental irritation. Chorea is commonly held to be a nervous manifestation of rheumatism. It certainly does occur more frequently in children giving a rheumatic history than otherwise. All of these manifestations of rheumatism in children show a tendency to occur singly, though they may co-exist. It is by reason of the mildness of the articular phenomena that so many cases are incorrectly diagnosed.

(*b*) **Chronic Articular Rheumatism.**—The term "chronic rheumatism" was formerly used to designate a large class of affections, many of which are now known to be of non-rheumatic character, and hence have been assigned to other categories. There still remain many cases of chronic articular disease to which we are obliged to apply the term for want of a better designation. It is probable that most of these are rheumatic, but, with increasing knowledge, the number of such cases must grow less.

Chronic articular rheumatism usually occurs in persons past middle life who have worked laboriously, and have been exposed to the vicissitudes of weather. It sometimes succeeds the acute inflammatory disorder.

The disease presents a variety of clinical types. In one, fortunately rare, the joint-lesions persist after an attack of rheumatic fever. In other words, there remains a chronic synovitis. The patient suffers from more or less pain, especially in damp weather, and eventually deformities appear. Heredity seems to be an important factor in producing these cases.

In the type more commonly observed, the patient complains of pain and stiffness of various joints. The extent of the suffering varies greatly from day to day, weather seeming to have a very important influence. Any number of joints may be involved. It is only exceptionally that articular swellings are noticeable. With few exceptions, the pains are worse in the early part of the day, and improve as the patient moves about. There is no necessary impairment of the general health.

(*c*) **Gout.**—The clinical features of typical gout include painful swelling of the metatarso-phalangeal joint of the great toe and a moderate degree of fever, and scanty, high-colored urine. The onset of the attack is usually sudden, the initial symptom being the joint-involvement. The affected part is red, swollen, and exceedingly tender to any inflammation. The symptoms tend to remit in severity during the day, but return again at night. The maximum intensity of the disease is usually reached at about the third or fourth day. Under proper dietetic precautions, the attack disappears in about ten days. Exceptionally, joints other than the great toes may be involved. In order of frequency, these include the ankles, insteps, fingers, wrists and knees.

The associated fever is always moderate. It bears a definite relation to the number of joints involved and the intensity of the local inflammatory changes.

The urine presents the characteristic features of febrile urine, *i.e.*, it is scanty, high-colored, and, on standing, it deposits a sediment of urates. Too much dependence must not be placed upon this symptom as evidence of the uric-acid origin of gout, for actual quantitative analysis demonstrates that uric-acid excretion during the early days of an attack of gout is diminished. With the crisis, the reverse condition obtains.

The subsequent history of the victim of gout is a variable one. Some few, more fortunate, never have a recurrence. In the majority, however, subsequent attacks appear at intervals of once or twice yearly. Sometimes one attack follows closely upon another ; or, again, it may happen that after a long period of suffering in this way the disease disappears, and gives the patient no further trouble for years.

The typical gout, above described, never offers any diagnostic difficulties. Even the etiology of the disease tends to explain the nature of these gouty paroxysms. As is well known, gout is an hereditary affection, tending to transmission from parent to child over many succeeding generations.

The dietetic habits of the individual tend to precipitate attacks, and maintain them when they appear. Particularly damaging to the gouty subject are excessive quantity of food, especially that of nitrogenous character, and indulgence in alcoholic beverages, such as port, sherry, Burgundy, ale, beer and champagne. Usually, gout attacks persons at or beyond middle life. Nevertheless, no age, not even childhood, is exempt.

Atypical gout presents clinical features which, at times, make their interpretation a very difficult matter. If, as is often the case, the patient with these irregular symptoms gives a history of one or more paroxysms of frank gout, the diagnosis is easy. But it is different when we have only the irregular symptoms upon which to base an opinion. It is the rule, however, for the irregular gout to appear in persons who have had repeated seizures, or who exhibit a remarkable tendency to the disease. The symptoms in these cases include eczema, pruritus, psoriasis ; affections of the eye, as conjunctivitis, iritis, or glaucoma ; of the nervous system, as neuritis or neuralgia ; of the vascular system, as palpitation, tachycardia, arrhythmia, angina pectoris, myocardial degeneration, and arterio-sclerosis ; of the kidneys, as chronic interstitial nephritis ; of the respiratory system, as asthma and recurrent bronchitis ; of the digestive system, as chronic pharyngitis and dyspepsia.

Notwithstanding statements to the contrary, none of these manifestations possess features by reason of which their gouty nature may be unerringly recognized. In cases in which other explanations are unsatisfactory, one may infer a gouty origin by reason of the habits of the individual, *i.e.*, partaking too freely of food and fermented liquors, and the leading of a sedentary life.

Retrocedent gout is an interesting, and, at times, a most dangerous variety. It is especially apt to occur during the course of an acute attack, the articular symptoms disappearing, and, at the same time, evidence of disease of the stomach or heart is presented. It is not unusual for such cases to prove fatal. Cerebral symptoms have thus been said to occur, and include headache, coma, or delirium.

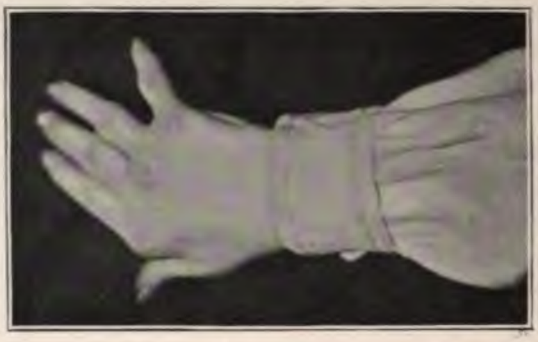
Chronic gout is generally the result of repeated attacks of the acute disorder. This leads to deposits of urates in the joints, with deformities of these structures. The feet first, and the hands later, are affected. Even the knees, elbows and tendons may be invaded. Not an uncommon symptom is the presence of tophi in the ears. Many clinicians rely upon the presence of the latter as an important diagnostic sign. The general health of the chronically gouty patient suffers considerably, presenting all the clinical phenomena of arterio-sclerosis and its organic complications.

(*d*) **Arthritis Deformans.**—Here we have an affection which has been unfortunate enough to receive several misleading designations. Of these, the one very commonly used is especially unfortunate, as it suggests that the disease is a combination of gout and rheumatism, while, in point of fact, its etiology has no relation to either of these affections. This name has, moreover, led to the introduction of incorrect if not positively pernicious methods of treatment. *Osteo-arthritis* is unobjectionable scientifically, but is not as convenient as arthritis deformans.

The clinical groupings of the symptoms of arthritis deformans are such as to make it easy to study the disease as appearing in various types, including the generalized or polyarticular, the mono-articular, and Heberden's nodes. The generalized or polyarticular forms may follow an acute or chronic course; nearly always, however, the latter.

The *acute* form of arthritis deformans occurs especially in children and young adults, and is characterized by inflammatory phenomena in the affected joints. The same erosions of cartilages and bones that appear in the chronic cases occur in the acute. The lesions generally commence in one of the smaller joints, especially in the metacarpo-phalangeal articulations, and extend rapidly to the larger ones. In this, as in other varieties, symmetry of articular involvement is the rule.

FIG. 178.



The hand of arthritis deformans in a girl aged 21 years.

The *chronic* form is far more frequent. It follows a slowly progressive course. It nearly always begins in the joints of the fingers, and involves all the joints of the body, producing deformities and immobilities in all the invaded parts. Even the maxillary joints and the spinal articulations are finally involved. The affected joints become swollen and distorted by reason of erosion of cartilages, eburnation of the ends of the long bones, and the formation of osteophytes. Finally the joints become immovable, not from true ankylosis, but by "locking" of the articular faces from the osteophytes and the articular changes. Marked atrophy of the muscles controlling the movements of the affected part are almost invariably present, and are due to the combined effects of non-use and ascending neuritis. Still later, these muscles become contractured, thus serving to increase the deformities. Finally, the changes invade the bursæ and tendons.

Pain is an inconstant feature. It occurs to a moderate degree in most cases. Exceptionally, it is sufficiently severe to demand special measures for its relief. In quite a number of instances it is entirely absent.

The constitutional disturbance is usually well-marked. This consists, as a rule, of a moderate degree of anæmia and general debility. Very early in the course of the disease, rapid action of the heart with or without palpitation appears. There may be associated a peculiar pigmentation of the skin, which has been designated "rheumatic." In the fully-developed disease the patient emaciates considerably, and this condition may give rise to considerable alarm, and lead to the suspicion that complications exist.

The mono-articular *variety* attacks by preference people of advanced years, especially men, and is very liable to result from traumatism. It usually affects one of the larger joints, as the knees, elbows, hips, or shoulders.

Heberden's nodes occur in the smaller joints of the hands and fingers. They consist of small, hard swellings in these locations, and are due to enlargement of the ends of the bones. At times, the affected parts are painful. As a rule, the disease does not extend to other portions of the body.

The causes of arthritis deformans include heredity, young adult life, the female sex, the neuropathic constitution, and defective hygienic surroundings.

(*e*) **Gonorrhœal Arthritis; Gonorrhœal Rheumatism.**—Gonorrhœal arthritis is a preferable designation for this disease, because it is a joint-inflammation dependent upon infection, which infection is presumably not that of the rheumatic poison. It may first manifest itself any time after the first week of the primary disorder, which need not necessarily be of the urethra. The larger joints are usually affected, the knees suffering more frequently than any of the others. The local symptoms include pain and swelling of the affected joints. The evidences of inflammation other than the intra-articular effusion are not prominent, for the affected parts are not hot to the touch, nor is there greatly increased redness. Important distinctions from acute inflammatory rheumatism are found in the absence of fever and the tendency of gonorrhœal arthritis to remain in the joints which it first attacks, and do not shift from part to part.

The disease ordinarily runs a favorable course, though, exceptionally, suppuration takes place, and this leads to permanent damage. Sometimes fibrous adhesions remain after subsidence of the disease. Once in awhile, gonorrhœal arthritis appears to act as the determining cause of a deforming polyarthritis.

The various fasciæ, especially those of the feet, become involved, and this leads to pain in the heels. Some cases are complicated by myalgia, especially of the lumbar region.

(*f*) **Scarlatinal Rheumatism.**—An apparently rheumatic inflammation of joints sometimes appears during the course of scarlatina. It is characterized by fever, swelling of the joints (usually the smaller) with intra-articular effusion, and pain. It is alleged to attack subjects who are predisposed to rheumatic fever, and exhibits the migratory character of that disease. Unlike true rheumatism, there is manifested at times a tendency to suppuration of the affected joints (*scarlatinal pyæmia*). It usually appears towards the end of the first week of scarlet fever, and its onset is favored by a severe attack of the primary disorder. Ordinarily it follows a favorable course, and is rarely succeeded by cardiac damage.

(*g*) **Simple Synovitis.**—In this connection, the term "simple synovitis" is used to designate a local joint-affection. It may occur as a simple serous inflammation of the synovial membrane, the inflammation may take on a purulent character, or there may be what is known as dry synovitis. *Synovitis with serous effusion* is generally the result of traumatism, and makes a good recovery in a week or ten days. *Suppurative synovitis* is a more serious disorder, and is due to infection. It may be that, from the first, the suppuration invades the joint-structures, and then we call it a *suppurative arthritis.* The latter affection is most dangerous to the future integrity of the affected joint, as necrosis of bone and cartilages is almost certain to take place. In all the varieties of synovitis we have the local symptoms of inflammation, the constitutional phenomena varying according to the severity of the local process and the character of the effusion.

(*h*) **Hæmophilia.**—The enlarged joints in connection with the hæmorrhagic tendency constitute the diagnostic features of hæmophilia. The inflammatory condition of the joints and the peri-articular tissues are believed to be dependent upon small hæmorrhagic extravasations. The swelling of the joints is ofttimes extreme. There is nearly always associated some discoloration of the overlying skin. Permanent changes in the affected joints may result.

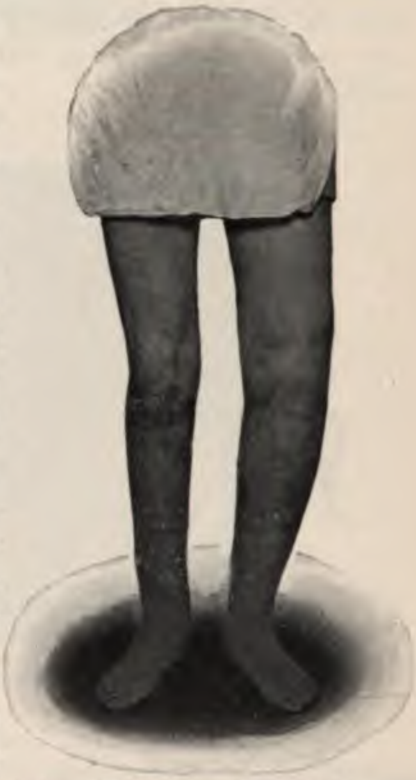
(*i*) **Syphilitic Disease.**—Syphilis is capable of producing quite a variety of joint-affections. These include simple arthralgia, monarthritis, polyarthritis, hydrarthrosis, disease of the cartilages, and disease of the bones. The diagnosis is based upon the history of syphilitic infection and the absence of other possible causes for the lesions, and, when undertaken before the onset of destructive changes, the benefit derived from the application of antisyphilitic measures.

(*j*) **Tuberculosis of Joints.**—While no age is exempt, children are especially liable to tuberculosis of joints. The articulations most frequently involved are the hip and knee. The bodies of the vertebræ are frequently the seat of tubercular caries. The general clinical manifestations of tubercular joint-diseases in general consist of chronicity, early impaired mobility of the joint, with atrophy of the muscles controlling the movements of the part, and tenderness on pressure.

(*k*) **Spinal Arthropathy.**—Joint-lesions are especially liable to occur in the course of locomotor ataxia and syringomyelia. The local changes may consist of a serous effusion, which is eventually absorbed, leaving the functions of the joint unimpaired, or it may progress to erosion of cartilages and bone-changes. The latter may consist of erosions; or irregular growths, sometimes of great size, may form in or about the joints. The erosion and absorption of the bony structures lead to dislocations under slight or even under no provocation. The deformities which may thus be occasioned are varied. The special feature of spinal arthropathies which should excite a suspicion of their true nature is the absence of fever and pain. Inasmuch as the majority of cases appear in the early stage of locomotor ataxia, they are very apt to consult the surgeon, who, being off his guard, treats them as examples of ordinary synovitis. Attention to the symptomatic totality will always demonstrate their spinal origin.

Occurring in syringomyelia, the history of the case and the dissociation of sensory phenomena should establish the diagnosis.

FIG. 179.



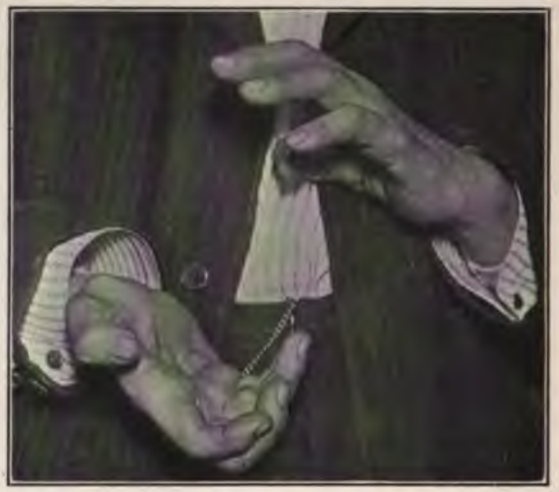
Arthropathy of left knee in a case of locomotor ataxia.

(*l*) **Rachitis.**—The so-called joint-swellings of rachitis consist, in reality, of enlargement of the epiphyses of the long bones at the wrists and ankles. These are manifested clinically as ring-like enlargements without external evidence of inflammation. They appear very early in the course of the disease,—even before there are any curvatures of the shafts of the bone,—and they disappear when the constitutional condition of the patient improves. Other parts than the wrists and ankles may be involved.

The Hand.—Certain alterations in the shape of the hand, as those observed in acromegaly, myxœdema, and pulmonary osteo-arthropathy, have already been described when speaking of the general characters of those affections. Other deformities, as Dupuytren's contraction, hammer-finger and trigger-finger, are mainly of surgical interest. There still remain several important changes in the outward appearance of the hand having important medical bearings. One of these, which has been a matter of note in medical cases for centuries, is the

Hippocratic Hand, or **Clubbed-Fingers.**—This condition is characterized by enlargement of the terminal phalanges, which become bulbous, giving the fingers an appearance which has not inaptly been compared to a "drumstick." The nails of the affected fingers are curved longitudinally. This state of affairs is brought about by all chronic conditions attended by enfeeblement of the circulation, including chronic heart disease, aortic aneurysm, pulmonary tuberculosis, chronic bronchitis, and empyema. It is due to a passive congestion of the parts resulting from their peripheral situation.

FIG. 180.



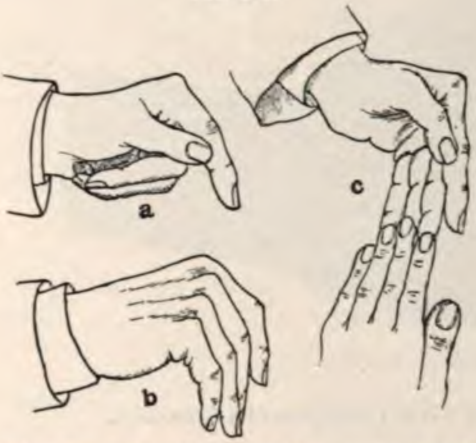
Claw-hand of progressive muscular atrophy.

The Claw-Hand.—The claw-hand, also known as the "*ape-hand,*" the "*maine en griffe,*" is observed in progressive muscular atrophy and ulnar neuritis. It is brought about by paralysis, with atrophy of the interosseous and lumbricales muscles. As a result, there occurs hyper-extension dorsally of the proximal phalanges, with flexion at the other phalangeal joints. The atrophy of the small muscles of the hand exaggerates the natural depressions between the metacarpal bones, and brings into prominence the flexor tendons. This condition occurs less commonly in neuritis of the median nerve, amyotrophic sclerosis, poliomyelitis anterior, and syringomyelia.

Paralysis agitans presents a deformity of the hand which is almost pathognomonic; if, indeed, any condition may be so designated. It is truly described as the "writing-hand," for the position is that assumed when grasping the pen, as in writing. There is a deviation of the hand towards the ulnar border of the forearm, and at the same time there is a coarse tremor, which was described by Charcot as a "pill-rolling movement." When this type of hand is not associated with tremor, as is frequently the case in the early stage of paralysis agitans, it may be confounded with the hand of arthritis deformans. It differs, however, from the latter affection in the absence of joint-enlargements, and in presenting other characteristic attitudes and deformities than those of shaking palsy.

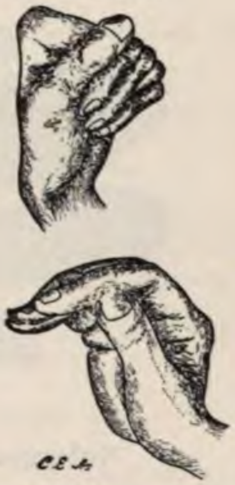
Dactylitis owes its medical interest to its relationship with tuberculosis and syphilis. Occurring as a manifestation of tuberculosis, it is dependent upon an osteomyelitis of the phalanges. The resulting changes produce a fusiform enlargement of the affected phalanges, the maximum diameter being about the middle of the bone. There is a strong tendency to spread to adjacent parts. The natural course of the affection is to caries and necrosis. Clinically, the objective symptoms usually attendant upon such bone-changes are present, and include redness, heat, and thickening of the soft parts. Tuberculous dactylitis is observed more frequently in childhood than during other periods of life, and usually in association with evidences of tuberculosis elsewhere.

FIG. 181.



Paralysis agitans. Posture of hands from contracture of the interossei: *a*, Left hand; *b*, Right hand. In the left hand the contracture is greater than in the other, so that the fingers cannot be extended even passively. The maximum passive extension is shown at *c*.—After Gowers.

FIG. 182.

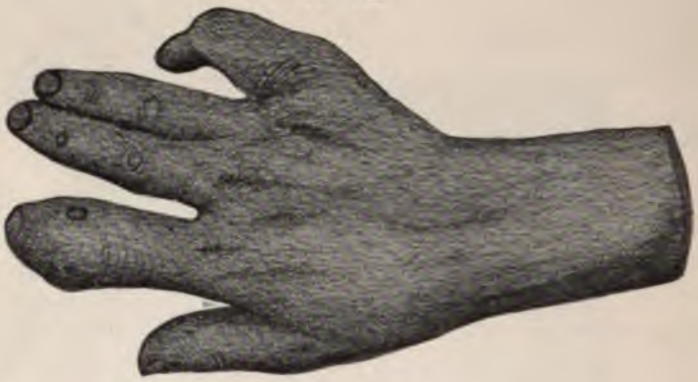


Contraction of fingers in paralysis agitans. Maximum voluntary extension.—After Gowers.

Syphilitic dactylitis is a manifestation of the tertiary and inherited forms of the disease. It is due to a gummatous infiltration of the bones or surrounding structures. When the ligamentous and subcutaneous structures are affected, the parts present a tense, hard or soft and semi-fluctuating swelling. The gummatous infiltration not infrequently breaks down, and the pathological process may extend to the joints and bones. It runs a painless course, and this enables us to distinguish it from arthritis deformans, gout, and whitlow.

More commonly, the gummatous process invades the phalangeal bones primarily. The process manifests itself clinically by a slowly progressive enlargement of the affected phalanx or phalanges, associated with evidences of inflammation, as pain and redness. The new tissue breaks down, ulceration and suppuration take place, and extensive disorganization of the parts results.

FIG. 183.



Syphilitic dactylitis.

Enlargement of the Lymphatic Glands.

Enlargement of the lymphatic glands may be local or general.

Local enlargements are due to infection within the area drained by the lymphatic vessels which are received by the enlarged glands. All infections, it must be remembered, are not equally active in this respect, and all persons are not equally susceptible. In some cases, the history of the causes producing such infection may not be forthcoming; or, the history being obtainable, the irritation seems to be entirely inadequate for the production of such results. The conditions existing in the mouth and throat are such that infectious processes are very liable to find their inception in these cavities. If lymphatic enlargement ensues, as it very frequently does, the affected glands are those of the neck and submaxillary region. In many instances the original infection, such as carious teeth, stomatitis, tonsillitis, and pharyngitis, is innocent from a prognostic standpoint. Many of the acute infections likewise produce these glandular tumors, especially measles, scarlatina, variola, and diphtheria. Malignant tumors are sooner or later attended by secondary involvement of lymphatic glands. Good examples of this are found in enlarged axillary lymphatics accompanying mammary cancer, and enlargement of the supraclavicular glands in malignant disease of the mediastinum.

Finding, then, lymphatic enlargements of limited distribution, it is important to know the areas to which their vessels are tributary. This is well stated in the following table, quoted from Ashby and Wright:

Distribution of the Lymphatic Glands and their Drainage Areas.

GLANDS.	HEAD AND NECK.
Suboccipital. Mastoid.	Drain posterior half of head.
Parotid.	Drain anterior half of head, orbits, nose, upper jaw, upper part of the pharynx.
Submaxillary.	Drain the lower gums, lower part of face, and front of mouth and tongue.
Suprahyoid.	Drain anterior part of tongue, chin, and lower lip.
Superficial cervical (lying beneath platysma).	Drain external ear, side of head and neck, and face.
Retropharyngeal.	Drain nasal fossæ and pharynx (upper part).
Deep cervical:	
Upper set along carotid sheath.	Drain mouth, tonsils, palate, lower part of pharynx, posterior part of tongue, nasal fossæ, parotid and submaxillary glands, interior of skull, and deep parts of head and neck.
Lower set in supraclavicular fossæ.	Drain upper set of lymph-glands, lower part of neck, and join axillary and mediastinal glands.
	UPPER EXTREMITY.
Supracondyloid.	Drain three inner fingers.
Axillary.	Drain upper extremity, dorsal and scapular regions, front and sides of trunk and breast.
	LOWER EXTREMITY.
Anterior tibial and popliteal.	Drain the deep lymphatics of the leg and receive some vessels from the skin of the leg and foot, chiefly on the outer side.
Inguinal:	
Femoral set (superficial).	Drain superficial vessels of the lower limb and partly of buttocks and genitals, and also perinæum.
Horizontal set (superficial).	Drain abdomen below the umbilicus, buttock, and genitals. The deep vessels of the lower limb go to the deep glands along the femoral vein.
Iliac.	Drain the pelvic viscera and the deep vessels of the genitals partly.
Lumbar.	Drain all the lower glands, uterus, ovaries, testes, kidneys.
Sacral.	Drain the rectum.

The principal constitutional causes of enlarged lymphatics are tuberculosis, syphilis, and lymphadenoma (Hodgkin's disease). The resulting enlargements may be limited to certain portions of the body, or they may be diffused. **Tubercular adenitis** is probably the most common of these.

In former years it was customary to look upon cases of this character as scrofulous. Now, we incline to the view that scrofulosis is, in most instances, identical with tuberculosis. The fact that most cases of so-called tubercular adenitis make a complete recovery does not militate against this view, for it is generally admitted that glandular involvement is the mildest manifestation of tuberculosis. The clinical features of a tubercular adenitis can hardly be said to be distinctive. In the majority of cases the glandular swellings are the only symptoms, which are not infrequently bilateral. They usually develop gradually. They may cease their progress for a time, and then once more progressively enlarge. They may be painful or tender, especially during those periods when they are increasing in size. Most of the cases sooner or later undergo suppuration. As to their relation to future tubercular processes, clinicians are undecided. It is recognized that but a small percentage subsequently develop pulmonary disease. The assertion that tubercular adenitis exerts a prophylactic influence against subsequent tubercular infection, although supported by some few high authorities, is most emphatically lacking the confirmation which should be received for so important a statement. Undoubtedly, in some instances, tubercular adenitis acts as a focus for general infection—sufficiently so, indeed, to call for great discrimination in their care and treatment.

Syphilitic Adenitis.—The lymphatic enlargements of syphilis may be studied as appearing in four classes:

1. Following closely upon the appearance of the initial sore, the lymphatics to which the affected part is tributary become enlarged. As the primary sore is upon the penis, it is the inguinal glands which are thus affected.

2. With general infection, numerous lymphatics throughout the body, but especially the supracondyloid and the posterior cervical, exhibit some degree of enlargement. The glandular tumors thus formed are rarely of any great size, and are freely movable beneath the skin.

3. In the late stages of syphilis, producing lesions of the bones, organs, etc., the lymphatic glands enlarge as they would from any other infection in the same area. Such lymphatic tumors, though syphilitic in one sense, are not such from a pathological standpoint.

4. Gummatous tumors of the lymphatic glands constitute the only form of great syphilitic enlargement of the lymphatic glands. The growth becomes intimately connected with the surrounding soft parts. It often undergoes softening in one or more places, and ultimately, if not treated, goes on to ulceration. It generally runs a slow course.

Hodgkin's disease is a disease characterized by general enlargement of the lymphatic glands, associated with weakness, emaciation, fever, anæmia, and enlargement of the spleen. The fundamental symptom is the glandular enlargement. Usually, this is manifested first in the lymph nodes of the neck, but it soon spreads and involves these structures in the axilla, groins, and other portions of the body. The tumors thus formed often grow to a remarkable size, and occurring about the internal organs, as in the mediastinum, produce serious but interesting phenomena by reason of pressure upon contiguous structures. The enlargements themselves are painless; they are hard, and they exhibit no tendency to break down and suppurate. The emaciation does not usually appear until the glandular involvement is prominent. Then it progresses with more or less rapidity, until it becomes extreme. The anæmia presents no characteristic features by reason of which the examination of the blood assists in the diagnosis. Hæmorrhages are not infrequent, especially from the nose. The fever usually comes on in paroxysms, which last for a time, and then disappears, only to return after a longer or shorter period. In some cases, however, it is continuously present. The associated enlargement of the spleen is usually not great. It is to be regarded as a valuable diagnostic sign in doubtful cases.

The diagnosis of Hodgkin's disease is always to be regarded as a difficult matter. Prominent authorities have stated that one should say "he diagnosed the case as one of Hodgkin's disease," and not that he "had seen a case of Hodgkin's disease." The conditions with which it may be confounded include a general glandular tuberculosis, sarcoma, carcinomatous lymphatic enlargements and leucocythæmia.

First, as to the differentiation of tuberculosis and Hodgkin's disease. When the physical signs and rational symptoms of pulmonary tuberculosis are present, there is not the slightest difficulty in establishing the true nature of the enlargements. But this is rarely the case. I can recall but one case of phthisis seen by me in which lymphatic tumors were present, and these suppurated early after their appearance. The family history and the family tendency of the patient, of course, count for considerable. In the early stages, the glandular enlargements of Hodgkin's disease are smooth, hard tumors, freely movable, and not adherent to adjacent parts. They increase in size gradually, remaining hard throughout, without any tendency whatever to destructive changes. The tubercular glands, on the other hand, are inclined to be tender to the touch, and ere long break down and suppurate. The inflammatory changes cause them to become adherent to each other, and thus they form one solid mass. Naturally, the skin overlying the tumor shows the discoloration incident to the inflammation beneath. Enlargement of the spleen appears early in Hodgkin's disease, and is an important diagnostic symptom.

From carcinomatous enlargements, Hodgkin's disease is to be differentiated by the stone-like hardness of the tumors in the former, and by the possibility of discovering symptoms which point to the presence of a primary tumor in the area tributary to the enlarged lymphatics.

The differentiation of malignant sarcoma of the lymphatic glands and Hodgkin's disease is regarded by high authority as next to impossible. The points upon which we may rely for their recognition have been stated by Bramwell as follows:

"The fact that the lymphatic enlargement is limited to one or more groups of glands which are in direct anatomical continuity; that the lymphatic swellings directly invade the surrounding tissues; that such organs as the mamma and testicles are affected; and especially that the lymphatic enlargements are secondary to a primary tumor in these or in some of the internal organs, and that the spleen is not enlarged, are in favor of true malignant sarcoma of the lymphatic glands, rather than of Hodgkin's disease.

"*Vice versâ*, widespread enlargement of several groups of glands in different portions of the body; the absence of a primary tumor; enlargement of the spleen; the early development of cachexia and implication of the tonsils and other organs in which lymphoid tissue normally abounds, are in favor of Hodgkin's disease.

"The differential diagnosis of Hodgkin's disease and of true malignant sarcoma is especially difficult,—in fact, impossible,—during life, in many cases in which the glandular enlargement is internal, *i.e.*, in which the bronchial and mediastinal glands are affected."

Enlargements About the Neck.

These include the enlargements of the thyroid gland, commonly known as *goitre*, and inflammation and swelling of the parotid and submaxillary glands (*mumps*).

An **enlargement of the thyroid gland** exhibits itself as a tumor situated at the level of the thyroid cartilages and upper end of the trachea, and rising with the larynx during the act of swallowing. Thyroid enlargements may indicate:

a. Parenchymatous goitre.
b. Cystic goitre.
c. Exophthalmic goitre.

Whatever may be the character of the goitre, it must be remembered that the enlargement is by no means necessarily uniformly distributed. It is not uncommon for one lobe alone to be involved, and it is usual for the brunt of the disease to be borne by one side. The isthmus of the thyroid may or may not be affected.

(*a*) **Parenchymatous Goitre.**—In this variety of goitre, the enlargement is smooth and firm, and may involve the whole or part of the thyroid gland. When of small size, there are no symptoms other than the enlargement. The goitre may, however, increase in size, and thereby produce numerous pressure phenomena, varying according to the structures thus affected. Thus, by pressure upon veins interference with the return circulation of the head takes place, as evidenced by turgescence of the face, headache and drowsiness. By pressure upon the arteries, cerebral anæmia is produced. Pressure upon the trachea interferes with breathing; and, lastly, there may appear a very interesting set of phenomena from compression of the laryngeal nerves.

(*b*) **Cystic goitre** produces the same pressure symptoms as the other varieties. The one objective symptom upon which we rely for the recognition of its cystic character is a sense of fluctuation in the tumor. In cases in which the walls of the sac are thick and firm, as not infrequently happens, fluctuation cannot be detected. Under such circumstances a diagnosis is possible only after an exploratory puncture.

(*c*) **Exophthalmic Goitre; Graves's Disease; Morbus Basedowii.**—There are three cardinal symptoms upon which we are accustomed to rely for a diagnosis of exophthalmic goitre. These are exophthalmos, rapid heart action (tachycardia), and enlargement of the thyroid gland. Characteristic as is this symptom group, it is possible for any one of these to be absent, and the case still be one of the disease under study. The tachycardia is now generally regarded as an essential clinical phenomena, without which the case cannot be one of exophthalmic goitre.

The enlargement of the thyroid gland usually appears gradually in association with the other symptoms. But it may appear rapidly in exceptional instances, and it may assert itself in advance of the other phenomena. As a rule, it does not reach the high grade observed in other varieties of goitre; indeed, it may at times be so slight as to require care for its discovery. The enlargement is evenly distributed over the entire gland, though it is not uncommon for one side to be somewhat larger than the other. To the touch it is firm and elastic, and pulsations are plainly evident. It is sometimes possible to discover a thrill over the goitre.

The exophthalmos may likewise be so slight in degree as to escape notice, and in some instances is noteworthy because of its absence. Like the goitre, it is usually gradual in onset, but may appear rapidly. It is nearly always equally marked in both eyes. When it happens that one side is more prominent than the other, it is usual for the corresponding side of the thyroid gland to be the larger. When the exophthalmos is fully developed, the characteristic staring expression of the face is produced. The eyes are wide open, and a ring of sclerotic is visible around the entire cornea. In extreme cases it is impossible for the eyelids to close over the protruding eyes, and the integrity of the eyeball is endangered. Attendant upon the exophthalmos are two other phenomena, which will be fully described in the section on eye symptoms, namely, *von Graefe's* and *Stellwag's signs*.

The prominent circulatory symptom is a remarkable increase in rapidity of heart action. No disease presents this phenomenon to such a degree. Ordinarily, the pulse-rate will be found to be about 120; but it is not so very uncommon to meet with examples of the disease in which it persists at from 180 to 200 per minute for days at a time. With this tachycardia, vascular tension is much diminished. The force of the heart-beats is apparently much increased. The heart is unduly irritable, slight emotional influences serving to excite palpitation or to increase the already rapid action. Murmurs may be present, but are not necessary symptoms. Dilatation is not unusual as a sequence of the perversion of heart function.

Aside from the cardinal symptoms, numerous other phenomena occur with sufficient frequency to be regarded as important phenomena of the disease. Prominent among these is the tremor, which is almost constantly present.

It is said by some that mental changes are commonly present, but this, according to my experience, is doubtful. Excessive sweating, diminished electrical resistance of the tissues, œdema, polyuria, amenorrhœa, and, in the male, impotence, constitute other symptoms of note.

Enlargements of the Parotid Gland.—Enlargement of the parotid gland may be the result of inflammation or of the formation of a tumor. Inflammation may be idiopathic or symptomatic. Idiopathic parotitis is commonly known as mumps. Symptomatic parotitis occurs as a complication of acute infectious diseases, notably croupous pneumonia, septicæmia, typhoid fever, typhus, small-pox, measles, etc.

Mumps.—This disease is known to be the result of contagion, the exact nature of which is not understood. It attacks children and young adults by preference. Infants appear to be almost entirely exempt. Its period of incubation is about two weeks. The victims of mumps are believed to be able to transmit the disease for a period beginning one day before the appearance of the swelling to two weeks after the entire subsidence of the fever. The initial symptom of the disease is fever, which is followed almost at once by pain and swelling in front of and below the ear on one side. The swelling increases rapidly, and may cause considerable distortion of adjacent tissues, even into the neck and cheek. The swelling is boggy to palpation,—never indurated. Stiffness and pain make movements of the jaw difficult, if not impossible. Thus, chewing and talking are greatly hampered. It is the rule for one side to be involved at first, the disease spreading to the other. Very exceptionally, both sides are affected simultaneously. Aural symptoms, consisting of earache and hardness of hearing, are sometimes present. In the course of a few days the swelling begins to subside, and in a week or ten days all symptoms will have disappeared. Mumps is, as a rule, an insignificant affection. Occasionally its course is interrupted by complications, of which orchitis in the male and mastitis in the female are most commonly observed. Exceptional complications include disease of the liver, lungs, eyes and ears, and nervous system.

Symptomatic Parotitis.—The onset of a parotitis during the course of one of the specific infectious fevers is characterized by a fresh accession of fever, or of aggravation of the febrile movement already present. Owing to the asthenic condition of the patient, subjective symptoms pointing to the parotid gland as a seat of inflammation are not prominent, so that the swelling is usually the first evidence of a local lesion. It is very common for the inflamed gland to undergo suppuration, though some cases end in resolution. If the resulting abscess is not evacuated promptly, serious consequences may ensue by reason of the burrowing of pus. It is always a serious affection, but it is to be dreaded much more when it occurs during the height of the primary disease than during the stage of convalescence.

Discolorations of the Skin.

A study of the many skin-lesions and the diseases of which they are suggestive involves the entire domain of dermatology, and hence is out of place in a work on general medical diagnosis. It will suffice our present purpose if only those alterations in the appearance of the skin having relation to general medical disorders be here considered. Symptoms bearing upon alterations in color may first be considered in this connection.

Color of the Skin.—Physiologically, the color of the skin shows important relation to age, occupation and habits, and to the individual. Infants exhibit delicate, soft, pale-pink skin. With increasing years this characteristic is lost. Finally, in old age, a muddy pale,—it might be called bordering on the sallow or ashy,—is manifested. This aging of the skin is influenced to a considerable extent by the individual's mode of life and his ability to resist the ravages of time. Persons of poor constitution exhibit early in life a cutaneous appearance which would be more appropriate in those many years their senior. Blondes, as a rule, preserve their youthful color to a much later period of life than do brunettes.

Exposure to wind and sun produces the well-known changes of "tan" and "sunburn." As to which of these changes is manifested, much will depend upon the idiosyncrasies of the individual; for some persons, under the influence of an out-of-door life, assume a browned appearance, while with others the skin takes on a bright redness. Of course, severe and acute exposure to the sun's rays will produce sunburn in any case. The severity of the phenomena will vary according to the susceptibility of the patient's skin.

The discolorations resulting from exposure to wind and storm, as exhibited in those following an out-of-door occupation, is well known to all. They may at times be so intense as to be suggestive of pathological changes.

Error from this cause may be safeguarded by comparison with the portions of the cutaneous surface covered by the clothing.

There is no normal standard of color for the skin. We are all aware of the many types of complexion, and the divisions of individuals into blondes and brunettes, and of the various grades of these. We know, too, that complexion types are prone to run in families. The pronounced "fairness" of the complexion of some individuals might well be suggestive of anæmia were it not that the normal redness of the mucous membranes indicated otherwise; and the dark, almost brown skin of pronounced brunettes arouses thoughts of a Latin-American extraction.

Pathologically, the skin may be

a. Abnormally pale (color decreased).
b. Abnormally red (color increased).
c. Discolored (alterations in color).

(*a*) **Pallor of the Skin.**—Pallor of the skin may be of short or long duration. *Evanescent pallor* is a common attendant upon depressing emotions and the onset of certain acute symptoms and illnesses. The readiness with which pallor appears under these circumstances depends in great measure upon the individual's idiosyncrasies. Thus very slight fright, grief, the receipt of unexpected bad news, is sufficient cause for the supervention of a pallor which may in some cases be well-described as "deathly." In the same way the skin may become pale and the surface of the body cold under the influence of syncope, abdominal pain, nausea, and the vascular neuroses. In the condition last-named the pallor is not infrequently localized, *e.g.*, to the arms and hands in the acro-neuroses (acroparæsthesiæ).

Pallor which comes on suddenly, and persists, especially if, as is practically always the case, it is attended by symptoms of collapse, is indicative of hæmorrhage. It thus constitutes an important indication of ruptured tubal pregnancy, ruptured aneurysm, intestinal or gastric perforation, and intra-gastric and intra-intestinal bleeding.

Pallor of gradual onset is certain evidence of bad health. In fact, it is an almost invariable attendant upon all chronic illnesses excepting those of the nervous system. When of a severe grade it is suggestive of one of the forms of anæmia, or of a diathetic disorder. In any event, it is the direct result of diminution in the coloring and corpuscular elements of the blood. The anæmias which are attended by the most pronounced pallor include chlorosis, pernicious anæmia and leucocythæmia. In chlorosis, the pallor is commonly described as possessed of a greenish hue; in pernicious anæmia, as of a lemon-yellow; it has also been described as a straw-color. In chronic Bright's disease the skin ofttimes assumes a waxy appearance, due to the combination of anæmia with œdema. In cancerous cachexia, the pallor presents a yellowish tinge. Notwithstanding the general acceptance of these characteristics of individual conditions, positive reliance must not be placed upon them as diagnostic. They are simply suggestive.

The pallor of general ill-health, as in tuberculosis, has no special characteristics. It has the same features as those presented by a run-down state from any cause, as from deficient aëration, malnutrition, etc.

(*b*) **Increased Redness of the Skin.**—A physiological hyperæmia of the face may occur in some individuals as the result of emotional causes, especially from anger. In the peculiarly sensitive it may give rise to positive discomfort, and may then be regarded as pathological.

In chronic ill-health the vaso-motor mechanism may be so unstable as to give rise to various circumscribed flushes under slight provocation. Thus we have the "blush" of the cheeks in some cases of phthisis (the hectic flush), the red cheeks of pneumonia, the flushed face of cerebral congestion, etc. Some individuals, by reason of their dilated capillaries and low vascular pressure, exhibit a constant florid complexion.

The skin may present changes in color by reason of general and local diseases. Of these, a **yellowish** discoloration is known as **jaundice,** and is dependent upon a staining of the tissue by bile. In mild cases, a yellowish tinge only is visible. In the more severe cases the discoloration is intense, and may in the aggravated cases assume a greenish hue. With the jaundiced hue of the surface of the body are associated other symptoms, some dependent upon tissue-staining, and others upon *cholæmia*. The mucous membranes throughout the body take on a yellowish stain. This is best shown in the eyes (the conjunctivæ), the white portion of which becomes more or less yellow, and, very early in the case, beneath the tongue. The staining of mucous membrane may be obscured because of the natural redness of the parts. In the case of the inner surface of the lips, this obstacle to the recognition of the symptom may be overcome by pressing a glass slide against the mucous membrane, thus rendering it anæmic, and enabling the yellowish discoloration to become evident. The various secretions and excretions of the body likewise become bile-stained. This is shown especially in the lachrymal secretion, the saliva and the urine. In obstructive jaundice the discharge of bile into the intestinal tract is prevented, and the fæces present a pale or ashy color. As a result of the cholæmia there is ofttimes a general itching of the skin, slow pulse (sometimes as slow as 30 per minute), delirium and coma. These latter symptoms are observed only in severe cases. In addition to the general pruritus without eruption, there may be, as additional skin symptoms, urticaria, boils, herpes and lichen; hæmorrhages may be associated with the different types of jaundice, not only from the mucous membranes, but also in the subcutaneous structures. The usual mental condition is one of depression.

Jaundice is to be regarded as a symptom, and not as a disease. A classification of the causes capable of producing it is not readily made; at least, all of the classifications thus far proposed have not been satisfactory. Perhaps a division of the cases into *obstructive* and *non-obstructive* jaundice is about as good as any. Certainly a division into *hepatogenous* and *hæmatogenous* is open to more criticism. Gioffredi and Semmola divide the cases into primary and secondary. Under the former heading are included (i) Catarrhal jaundice; (ii) febrile jaundice; (iii) icterus gravis; (iv) emotional jaundice; (v) jaundice of the new-born. Under secondary cases are included (i) Jaundice due to infections; (ii) jaundice due to hepatic disease; (iii) jaundice dependent upon intrinsic and extrinsic stenosis of the bile-ducts.

Cases of obstructive jaundice may be further divided, according to the seat of the obstruction, into two classes: (i) In which the obstruction is within the tubes, *i.e.*, dependent upon swelling of the walls of the bile-ducts or the presence of foreign bodies within their lumen; and (ii) obstruction from without, as from pressure of tumors or enlarged viscera. The most important of the conditions to be included under the first class are catarrhal jaundice and obstruction of the common duct by gall-stones.

But there are some who even object to this simple classification, for they claim that biliary obstruction is incapable of producing jaundice unless accompanied by disturbance in function or alteration of structure of the hepatic cells.

Catarrhal Jaundice.—This is dependent upon a catarrhal inflammation of the larger bile-ducts, which finds its origin in an extension of an inflammation of the mucous membrane of the duodenum. Hence, the disease is generally preceded by symptoms of gastro-intestinal indigestion. These include loss of appetite, nausea, vomiting, constipation or diarrhœa. Then appears the jaundiced hue of the skin. Usually but moderate in degree, the discoloration never proceeds to the olive-green hue. Such symptoms as pruritus, slow pulse, xanthopsia, etc., are commonly present. The stools are of ashy hue; the urine is bile-stained. The liver is enlarged, sometimes to the extent of two or three finger-breadths below the normal. There may be some local pain; usually the liver is tender on pressure. Owing to the obstruction of the common duct, the bile is dammed up in the gall-bladder, which becomes distended. Catarrhal jaundice usually runs a course of two or three weeks. The first evidence that the obstruction has been relieved is found in the return of bile to the intestines, as shown by the dark color of the stools. A considerable time elapses, however, before the last remnant of bile-staining of the tissue disappears.

The diagnosis of catarrhal jaundice is based upon the appearance of jaundice after a period of gastro-duodenal indigestion. The absence of bile from the fæces is an important diagnostic symptom. It is also important that the physician exclude the possibility of the existence of any of the other diseases of the liver capable of giving rise to jaundice, especially of cholelithiasis.

Although jaundice is a frequent symptom of *gall-stones*, it is so often absent as to lose all value as a diagnostic symptom. It can never arise from this cause unless the calculus is obstructing the common duct, in which case symptoms of far greater importance will be present.

Some few cases of *jaundice of the new-born* are obstructive. The lesion in these cases consists of an occlusion of the common duct by a plug of mucus (Virchow). In such cases the symptoms disappear in a week or two. Other cases are undoubtedly dependent upon greater destruction of blood-corpuscles than can be taken care of by the liver. These likewise offer a favorable prognosis. Serious and even fatal causes of this condition in the new-born are absence of the hepatic or common duct, hepatic syphilis, and septic inflammation of the umbilical vein.

The various lesions which, by exerting pressure upon the duct from without, and thus obstructing its lumen, include aneurysm of the aorta, and hepatic or mesenteric arteries, cancer of the liver (the jaundice in this case being painless), malignant disease of the pancreas or right kidney, hepatic abscess, lymphadenoma, pregnancy (occasionally), and abdominal tumors generally.

Cases of non-obstructive jaundice are commonly spoken of as toxæmic or hæmatogenous. They include those of malarial fevers, acute yellow atrophy of the liver, pyæmic affections, pernicious fevers, yellow fever, Weil's disease, snake poisons, and poisoning by phosphorus, antimony, copper and mercury.

Acute Febrile Jaundice.—Under this title have been included several affections which, in the present state of our knowledge, cannot be differentiated. For the present, then, the term acute febrile jaundice must be regarded as being generic. Clinically, we may easily recognize benign and grave cases. There is an intermediate group presenting features simulating the extremes. The symptoms of acute febrile jaundice vary greatly, according to the severity of the case. Possibly it would be better to say according to the severity and nature of the infection or intoxication. It is ushered in with prodromal symptoms of a general character, but the important feature is fever, which, in severe cases, may attain the height of 104° F. There may also be epistaxis, intestinal hæmorrhages, and great prostration. The jaundice is one of the earlier features. Biliary coloring-matter is found in the urine; the stools, however, contain the usual quantity of bile. Physical examination shows moderate enlargement of the liver. In mild cases the fever disappears with the outbreak of the jaundice. In the benign cases there are no symptoms of cholæmia, hence the general condition of the patient is good. Such cases make a recovery in the course of two or three weeks. In the grave cases, hæmorrhages become a prominent symptom, delirium sets in, and death follows.

In the early stages of the disease the urinary secretion and the urea percentage are diminished. With improvement of the patient the quantity of urine becomes increased, and the urea percentage is above normal. Chauffard has referred to these phenomena as *polyuric* and *nitrogenous crises.*

Weil's disease seems to be but a type of acute febrile jaundice. It is characterized by a febrile icterus exhibiting a tendency to relapse.

Emotional Icterus.—The nature of jaundice arising from emotional causes is an unsolved problem. As a matter of observation, it may be stated that the jaundice appears within a very short time after the exciting cause. It is not attended by gastro-intestinal disturbances, and there is no alteration in the color of the stools. Most cases recover in the course of four or five days.

Bronze or brown discoloration of the skin, diffuse or in patches, may be observed in Addison's disease (of which it is the important diagnostic symptom), arsenical poisoning, diabetes, exophthalmic goitre, melanotic liver, pregnancy, syphilis, freckles and rheumatoid arthritis.

Addison's Disease.—The prominent clinical features of Addison's disease are two in number, namely, the generalized bronzing of the skin and the progressive asthenia. The discoloration of the skin is usually the first symptom noticed. Occasionally it is deferred until the disease is well advanced. In intensity it may vary all the way from a brownish-yellow to a deep bronze, bordering almost on the black. It appears first by preference on parts of the body which are apt to take on pigment under other circumstances, as the nipples, the genitals, and parts exposed to pressure and friction of the clothing. When the pigmentation occurs in patches, there is no well-defined line of demarcation. The mucous membrane of the mouth exhibits dark-bluish patches, to which reference was made when speaking of symptoms of the mouth. The asthenia, however, is the symptom which gives the patient the most concern, and the one demanding relief. Fainting attacks are common ; indeed, notwithstanding the good outward appearance of the patient, he may be obliged to keep in bed from sheer exhaustion. The heart's action is in keeping with the general weakness. Death usually results from the exhaustion, the patient dying in delirium.

The prognosis of Addison's disease is invariably unfavorable. A fatal result generally follows within a year, though life has exceptionally been prolonged for a number of years. Patients with Addison's disease are especially susceptible to tubercular infection.

Arsenical Poisoning.—Arsenic, when given medicinally for a prolonged period, is very liable to produce pigmentation of the skin. Especially is this accident liable to happen in cases of chorea, pernicious anæmia, and other diseases which are now treated, according to orthodox methods, by the administration of arsenic to the verge of tolerance. The history of the case establishes the diagnosis.

Pregnancy.—Pigmentation during pregnancy is observed especially about the nipples and along the linea alba, and about the genitalia. It is regarded as one of the symptoms upon which a diagnosis of pregnancy may be based.

Syphilis.—A persistent pigmentation of the skin in parts which have been the seat of eruptions or ulcerations is one of the features of syphilis.

Vagabond's Disease.—This is a condition described by Greenhow, and consists of a pigmentation of the skin through result of exposure to the sun and vicissitudes of weather, and the irritation of vermin and dirt.

Symptomatic Pigmentation of the Skin.—A number of diseases presenting well-defined symptomatologies may be attended by pigmentation of the skin, especially in blotches. Among these diseases are exophthalmic goitre, arthritis deformans, and melanosarcoma.

Of the eruptions of the skin, the province of the present work precludes the possibility of considering only those of the exanthemata, namely, measles, scarlatina, rötheln and variola. The eruptions of these diseases have been fully studied in the paragraphs dealing with these several affections, and to them the reader is referred for information.

CHAPTER XVIII.

THE NERVOUS SYSTEM.

It is in the clinical investigation of diseases of the nervous system that the careful weighing of all the symptoms, together with a consideration of all facts pertaining to the medical history of the patient, will be found especially important. Here, more than in any other class of affections, it is difficult to make diagnoses by empirical methods. It is essential that each individual phenomenon of the disease under investigation be carefully analyzed. Unfortunately, physical examination, valuable as it is, aids us less in the study of nervous than of any other class of diseases. We must depend, in very many instances, upon the symptoms as given us by the patient. Fortunately, these symptoms are usually presented in groups having well-marked associations, so that the experienced are not likely to be deceived by inaccuracies.

Diseases of the nervous system include organic and functional derangements of

a. The brain.
b. The spinal cord.
c. The peripheral nerves (including the cranial and spinal nerves).
d. Any two or all three of the above.

Disease of these may produce disturbance of functions relating to

a. The mind.
b. Motor power.
c. Sensation.

Clinical Study of Motor Symptoms.

The motor functions may be disturbed in the direction of excess, diminution, or alteration.

I. Under **diminution of motor power** we include paralysis, which may be localized or general, partial or complete.

II. Under **excess**, convulsions, spasms, contractures, tremor, tic, chorea, fibrillation, and associated movements.

III. Under **alteration**, disturbances of co-ordination, *e.g.*, ataxia.

Paralysis.—By this term is meant the loss of the ability to move a part by reason of a lesion of the motor nervous apparatus. Inability to move a limb because of anchylosis or inflammation of joints, local inflammatory conditions, or painful affections of any kind, must not be regarded as paralysis, for in these the power of voluntarily contracting the muscles is unimpaired; but the patient refrains from exerting it because of the inutility of the effort, or because of the pain occasioned thereby. It may seem unnecessary to impress upon the reader the possibility of mistaking these conditions interfering with motor functions, but I can assure him that it is by no means uncommon for patients to state an inability to move a part, designating the loss of function as "paralysis," the physician not discovering the error until he proceeds with his physical examination of the diseased part.

The term **paresis** was formerly much in vogue to indicate a loss of power not amounting to absolute paralysis. It is an unfortunate one, inasmuch as it is in common use as applied to a form of insanity known as "*general paralysis of the insane.*" Owing to the liability to confusion, it is better to speak of "*partial*" or "*incomplete paralysis*," or of "*a paretic condition.*"

Another use for the term "paresis" has been found in a condition of muscular exhaustion. The muscle is capable of acting apparently with its normal vigor for a limited time, but soon becomes exhausted. Such a condition is not uncommon during the early stages of many chronic organic affections of the nervous system. Thus I have noted a tiring readily on walking, in progressive bulbar paralysis, at a time in the course of the disease when it was believed that the patient was simply "run-down," and "could walk readily enough when starting off, but soon exhibited irregularities in gait" in paraplegias of various types.

In every case of paralysis the following points should be investigated:

The mode of onset of the paralysis, with the associated symptoms.
The distribution of the paralysis.
Its association with rigidities, muscular atrophy, and sensory disturbances.
The electrical reactions of the paralyzed muscles.

According to its distribution, paralysis is designated

Hemiplegia, when it involves one lateral half of the body.

Paraplegia, when it involves the lower extremities.

Hemiparaplegia, when it involves one lower extremity.

Monoplegia, when it is limited to one limb or to a group of muscles.

Multiple, when involving two or more dissociated parts.

Alternate hemiplegia, when it involves the leg and arm of one side and the face or eye-muscles on the other.

The term **diplegia** in its strictest sense means a paralysis of two extremities without involvement of the muscles of the trunk. Clinically, it is hardly ever used with this meaning. Thus we speak of a "**cerebral diplegia**" referring to a double hemiplegia; **diplegia brachialis,** meaning a paralysis of both arms; and "**diplegia facialis,**" meaning a paralysis of both sides of the face.

The clinical investigation of paralysis includes an inspection of the affected parts while at rest or during attempts at voluntary motion; the resistance by the patient of passive movements made by the physician; and the use of dynamometers.

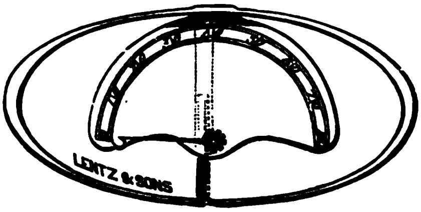
Simple inspection with the affected parts at rest is often sufficient to discover the presence of paralysis of the muscles of the face and eye.

Inspection of the paralyzed parts during attempts at voluntary motion is useful in loss of power of any portion of the body, but is especially important in cases with paralysis of the lower extremities when the patient attempts to walk.

The ability of the patient to resist passive movements gives important information as to the condition of the muscles moving certain joints, but especially the knees, hips, elbows and shoulders.

The **dynamometer** is especially applicable in determining the motor power of the hands. Modifications of the instrument may be employed in determining the existence of paralysis in numerous other portions of the body. Dynamometers are merely a convenience, not a necessity, in the recognition of loss of power. Their especial value is found in enabling one to make comparisons of the strength of paralyzed muscles from time to time. The one in most common use is that illustrated in the cut (Fig. 184). It is used solely for estimating the grasp of the hand. Many of these instruments are furnished with inner and outer scales. To place any definite value on the readings of dynamometers is liable to lead to error, as these instruments are not accurately calibrated. To say, therefore, that the patient is capable of exerting so many pounds of force, as shown by the dynamometer, is wrong, while it is perfectly admissible to say that he is capable of exerting that many degrees of force. The readings of dynamometers varying thus, physicians are obliged to learn to appreciate the value of the instruments to which they have been accustomed.

FIG. 184.



Dynamometer.

There can hardly be said to exist a normal standard of strength. In all cases one must be guided by comparing healthy with diseased parts. I find that, with the dynamometer which I use, healthy individuals are capable of registering all the way from fifty-five to one hundred and thirty-five degrees of force,—reading by the outer scale. The grasps of the two hands are practically always found to differ. The right hand is usually the stronger by from ten to fifteen degrees. In ambidextrous individuals there may be no such differences, and in those actually left-handed the balance of power is in favor of that side. In using the dynamometer, care must be observed that only the grasping power of the hand is exercised. The patient must not be permitted to lay the hand on some support or to swing the arm, in order to gain power. For the purpose of estimating the strength of the calf-muscles, the peronei, the anterior tibial group, the quadriceps extensors, the biceps, and numerous other muscles, special forms of mechanical contrivances have been designed. They are useful merely for the purpose of making clinical records complete. The most practical method of estimating loss of power in individual muscles is by resistance to passive motion. For example, to estimate the strength of the biceps, the physician extends the arm at the elbow, while the patient resists.

The determination of the presence of paralysis of one or both lower extremities, excepting in those in which the loss of power is absolute and permits of no motion whatever in the affected part, is best made by **inspection of the patient during the act of walking.** Not only may the presence of paralysis be thus discovered, but peculiarities in the gait often determine the variety.

In **hemiplegia** the affected leg is dragged after the other one has advanced, or is swung around in a semicircle when advanced. The latter applies to hemiplegia of organic origin only, for in **hysterical hemiplegia** the affected extremity is dragged directly forwards.

In **paraplegia** the gait will vary according to the associated phenomena. When associated with rigidity of the paralyzed parts (**spastic paraplegia**), the limbs move as in one piece; the toes scrape the ground; owing to the adductor spasm, the limbs are drawn in front of each other, or the toe of the advancing foot catches in the heel of the one in front. This gait is sometimes referred to as "**cross-legged progression.**" A variety of this gait is the "hopping gait," in which, because of the tension on the tendo-Achilles when the weight of the body is sustained by it, there occur clonic spasms of the calf-muscles (ankle-clonus).

When paraplegia is associated with flaccidity of the affected muscles, as in acute poliomyelitis anterior, progressive muscular atrophy, and multiple neuritis, peculiarities varying with the distribution of the paralysis and the associated deformities will be observed. If, as in multiple neuritis from alcoholic excesses, there is weakness or loss of power of the anterior tibial group of muscles, the patient is unable to perform dorsal flexion of the feet; hence, when walking, the feet droop, and the patient, in his efforts to make them clear the ground, lifts them high by making unusual flexion of the knee and hip. In other words, it is a **high-stepping gait with dropped feet.**

is brought into a position of dorsal flexion, and this position it maintains until it touches the floor again, which it does heel first, the sole of the heel following with a sort of thump. All the movements of locomotion are of a jerking character. Sometimes, in making a step, the patient lifts the foot much higher than necessary, not being able to judge distances correctly.

In *Friedreich's ataxia* the inco-ordination is, in part, like that of locomotor ataxia as above described, and in part a reeling motion, like that of alcoholic intoxication.

In *cerebellar disease* we have an inco-ordination which is strictly of a reeling character.

In the remarks that will follow, frequent reference will be made to the various reflexes. It is in order, therefore, to digress from our present subject,—paralysis,—and describe these phenomena, and give in some detail the lessons to be derived from their alterations in disease.

FIG. 185.

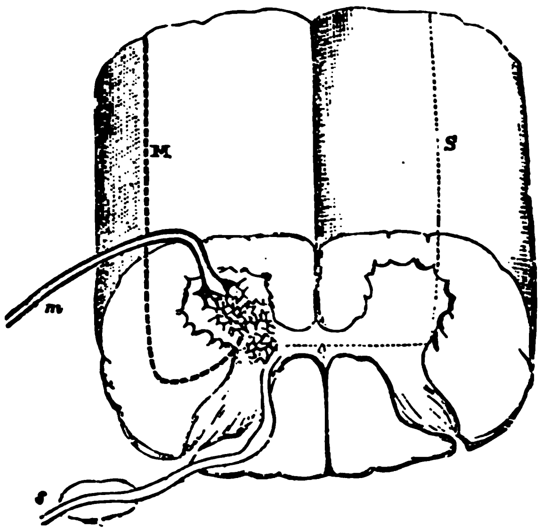


Diagram of a reflex loop. *M*, Motor, and *S*, Sensory pathways.

The Reflexes.—An examination of the various reflexes is a very important routine measure in all cases.

The reflexes are divided into

1. The *superficial* or *cutaneous reflexes*, and
2. The *deep* or *tendinous reflexes.*

The superficial reflexes are certain reflex movements produced in response to irritation excited in definite areas of the body. The value of the reflexes lies in the fact that their presence proves conclusively that the integrity of the corresponding reflex arc is intact. This reflex arc, as illustrated in the accompanying figure, consists of the motor and sensory fibres of the peripheral nerve, the anterior and posterior nerve-roots, the anterior cornua, and the posterior root zones. Any break of continuity in this path destroys the integrity of the arc, and the reflex movement is prevented when the proper irritation is applied.

The **superficial reflexes** are as follows: Plantar, gluteal, cremaster, abdominal, epigastric, scapular, and erector spinæ.

The **plantar reflex** is excited by irritation of the sole of the foot, which produces movements of the foot and toes. Its presence depends upon the integrity of the reflex arcs from the fifth lumbar to the second sacral segments.

Of late much attention has been paid to a modification of the plantar reflex, known as **Babinski's sign.** When, in infants, irritation is applied to the sole of the feet, there is dorsal flexion of the toes. In adults the same irritation excites plantar flexion of the same parts. When, in the case of organic disease of the lateral tracts, there is increase of the superficial reflexes,—the flexion of the toes in response to irritation of the soles of the feet,—the resulting movement of the toes is dorsal flexion; in other words, the reflex reverts to the infantile type.

The **gluteal reflex** is produced by irritation of the skin over the buttock, which produces contraction of the glutei. It depends upon the integrity of the arcs from the fourth and fifth lumbar segments.

The **cremaster reflex** is produced by irritation of the skin on the inside of the thighs. The resulting movement is sudden retraction of the testicle on the corresponding side. It depends upon the integrity of the first to the third lumbar segments. Care must be taken, in examining this reflex, not to confuse with it the vermicular movements of the dartos. When well-marked, the cremaster reflex may be excited by slight irritation of the outer or anterior surface of the thigh.

The **abdominal reflex** is produced by irritation of the skin over the side of the abdomen on a line perpendicular to the nipple. The resulting movement is a contraction of the rectus muscle of the corresponding side. It depends upon the integrity of the eighth to the twelfth dorsal segments.

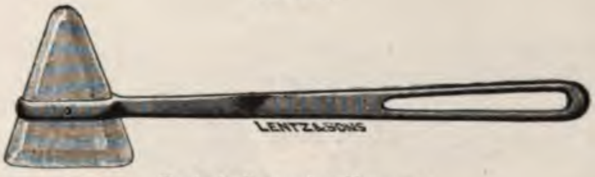
The **epigastric reflex** is produced by irritation of the side of the thorax on the line of the nipple. The resulting movement is a dimpling of the epigastrium. Its existence depends upon the integrity of the reflex loops from the fourth to the eighth dorsal segments.

The **scapular reflex** is produced by irritation of the skin between the scapulæ. The resulting movement is contraction of certain of the scapular muscles. It depends upon the integrity of the reflex loops of the fifth cervical to the first dorsal segments.

The **erector spinæ reflex** is produced by irritation of the skin on either side of the spinous processes.

The tendon reflexes of practical value are the *knee-jerk*, the *ankle-clonus*, and the *triceps-jerk*. A tendon reflex phenomenon is possible wherever there is a tendon that can be put on the stretch and a blow applied to it. Of the tendon reflexes, the one known as the **knee-jerk** is the most important. A proper understanding of the methods by which it is elicited is in order. The method most commonly in use is the following: The patient should be seated. The knees should be bared. Then the leg to be examined should be thrown over its fellow, and allowed to hang as if lifeless, so that no involuntary rigidity shall be permitted to interfere with the reaction. Then with the outer edge of the hand, or with a percussion hammer, or the edge of the ear-piece of a stethoscope, a smart blow should be applied to the ligamentum patellæ, just below its insertion into the patella; when in health, a vigorous contraction of the quadriceps extensor ensues, and the foot is jerked suddenly upwards. In applying the test, baring of the knees is absolutely necessary for the sake of accuracy in observation; especially is this so in cases in which the blow applied through the clothing has failed to elicit a response. It cannot be conclusively stated that the patellar reflex is absent in any given case unless this be done. No less important is the injunction that the limb examined be held perfectly limp and lifeless. Frequently have I seen cases in which the patellar tendon reflex had been declared to be absent simply because the precaution of securing perfect relaxation of the limb had been neglected. There are cases in which this is a difficult matter, owing to the inability of the patient to fix his attention away from the operator's movements. In such it has been my custom to adopt the device first suggested by Jendrassik, to semi-flex the fingers, hook them together, and exert strong traction. While this is being done, the blow is applied to the ligamentum patellæ as before, when, if the knee-jerk be present, it is readily elicited. Any other voluntary movement made by the patient will secure the same object. Thus, asking the patient to count aloud, or to look into vacancy and wink rapidly, and while the physician applies the blow to the ligamentum patellæ, will secure a response as readily as by the method of Jendrassik.

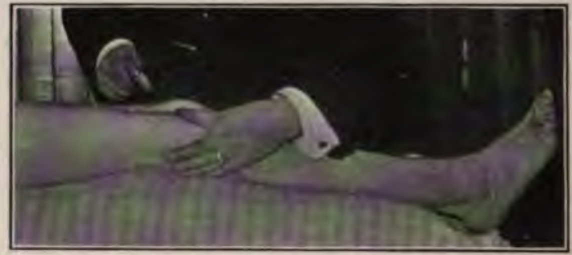
FIG. 186.



Taylor's Percussion Hammer.

In very stout persons the above method of applying the patellar reflex is impracticable, owing to their inability to sit with legs crossed. In such cases the patient should be directed to sit on the edge of a table or desk, with the legs dangling over. A blow applied to the ligamentum patellæ, as in the previously-described method, produces the knee-jerk.

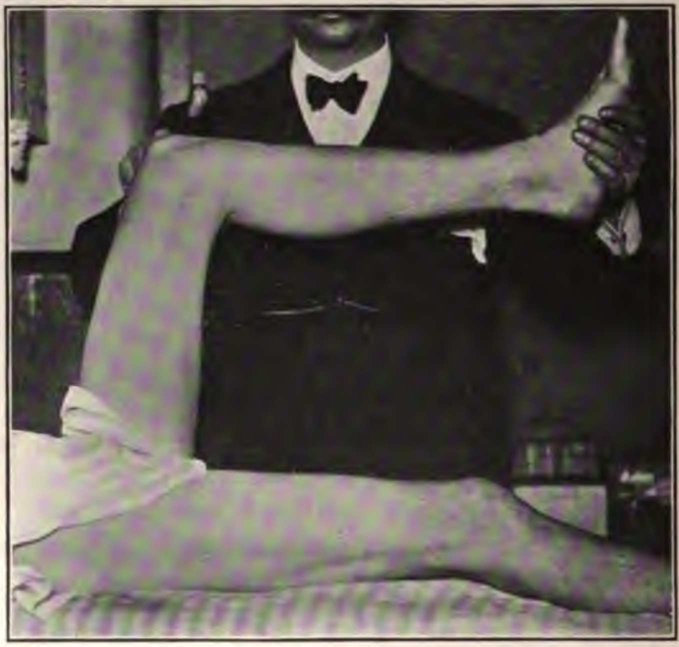
FIG. 187.



A method of obtaining the knee-jerk with the patient in bed.

Cases will frequently occur in the practice of all of us in which the patient's illness is of such a nature as to make it impossible to get him out of bed to apply one of the above-described tests. We may then resort to the following method, first suggested by Dr. Angel Money. The patient lying on his back, the thigh is flexed at right angles with the body, and

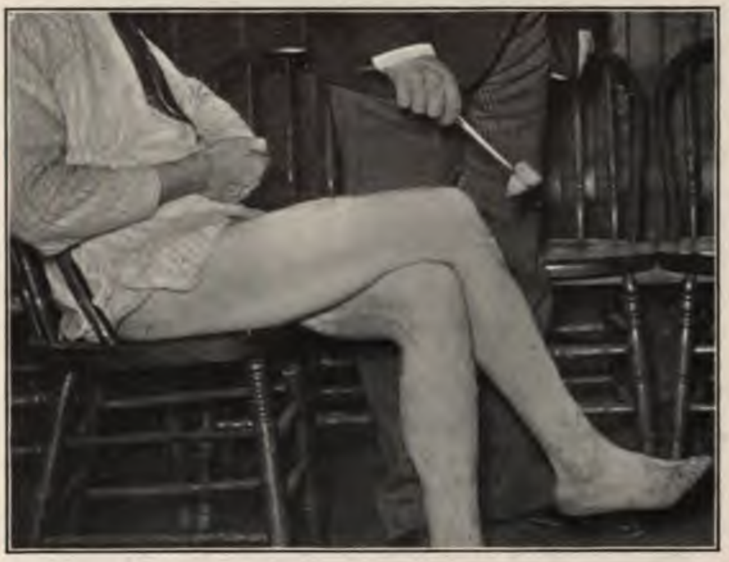
FIG. 188.



Money's method of obtaining knee-jerk with patient in bed.

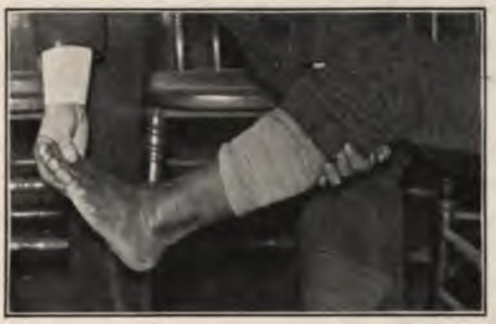
the knee at nearly a right angle with the thigh; the foot is then supported loosely in the palm of the operator's hand, as in a stirrup, care being taken that the patient exert no effort to maintain the limb in this position. A blow is then applied to the ligamentum patellæ, as in the other methods of examination; the knee-jerk then takes place.

FIG. 189.



Obtaining the knee-jerk according to method of Jendrassik.

FIG. 190.



Obtaining the ankle-clonus.

The **ankle-clonus** is produced as follows: The examiner takes the patient's leg, supporting the heel on his knees and the popliteal space with the left hand, observing care that the limb is slightly flexed at the knee-joint. Then with the right hand applied to the ball of the foot, that member is pushed suddenly upward, maintaining the foot in dorsal flexion, and putting the tendo-Achilles on a stretch. If the ankle-clonus is present, there appears a rhythmical movement of the foot, which, in case the phenomenon is at all well-marked, continues for several minutes, or until the pressure is removed. Unlike the patellar reflex, the ankle-clonus is a pathological condition.

A **spurious ankle-clonus** is sometimes observed. It must not be confounded with the true clonus above described. It consists of a few abortive and irregular oscillations. It is found in hysteria, neurasthenia and malingering.

The **front-tap contraction** is a delicate test of increased myotatic irritability. To produce it, the leg is extended at the knee, tension is brought to bear upon the tendo-Achilles, and a blow is applied to the tibia. If it is present, there ensues a jerk produced by contraction of the calf-muscles.

Westphal's paradoxical contraction is the persistent spasm of a muscle taking place when its origin and insertion are suddenly approximated. It is best produced in the peroneal muscles by sudden dorsal flexion of the foot. It is observed in functional nervous diseases.

Tendon reflexes may also be obtained in other parts of the body than the legs. Those of the head include the **jaw-jerk** and **Chvostek's sign.** To elicit the jaw-jerk, the patient is directed to hold his mouth partly open, and, as far as possible, relaxed. A tongue-depressor or spoon-handle is placed upon the teeth of the lower jaw with slight pressure, and a sharp blow is applied to it with a percussion hammer. This will cause a quick though slight upward jerk of the lower jaw. The absence of this reflex is without much clinical significance, as it is by no means always present in health. It is apt to be excessive in neurasthenia and hysteria, and sometimes in patients with profound coma.

Chvostek's sign occurs only in tetany. It is really a periosteal reflex. To produce it, the zygomatic arch just in front of the ear should be tapped with a percussion hammer, and then will follow a series of sudden twitchings of the face, especially of the elevators of the angles of the lips and the muscles of the eyelids. It may also be elicited by striking over the malar bone or the infra-orbital foramen.

In the arms, the important reflexes are those referred to the triceps, biceps, and supinator tendons.

The **triceps-jerk** is produced as follows: The patient sits with arm flexed at somewhat of an obtuse angle at the elbow, the forearm resting lightly on the lap. A blow, applied by means of the percussion hammer to the triceps tendon, produces the elbow-jerk.

To obtain the **biceps reflex,** the arm is flexed at a right angle, the forearm is rested upon anything which may happen to be convenient, the left thumb of the observer is then placed upon the biceps tendon, and a blow applied with the percussion hammer. A distinct jerk of the forearm will ensue in healthy individuals.

To obtain the **supinator reflex**, a blow is struck on the radius, just above its styloid process.

In cases of paralysis of arm with marked spasticity, it is possible to obtain a **wrist-clonus** by either dorsal flexion or extension at the wrist, and holding the hand in the forced position.

The reflexes are absent in any lesion impairing the integrity of the reflex above described. We therefore find them absent in

Nerve injuries.
Neuritic paralysis.
Lesions of the spinal nerve-roots.
Acute anterior poliomyelitis.

The tendon reflexes are absent in the above, and, in addition, in

Locomotor ataxia.
Cerebellar disease, sometimes.
Meningitis, sometimes.
In the earliest stages of apoplectic extravasations.

The cutaneous reflexes are absent or diminished and the tendon reflexes exaggerated in

Cerebral lesions.

Both deep and superficial reflexes are exaggerated in

Spinal-cord lesions involving the lateral tracts of the cord, hence in spastic paraplegia, ataxic paraplegia, disseminated sclerosis, some cases of tumor of the cord, and pachymeningitis from Pott's disease.
In neurasthenic and hysterical conditions.
Exhausting diseases.

Myoidema.—Very frequently, when practising percussion of the chest in persons suffering from phthisis and other wasting affections, we observe contractions of individual muscular strands in response to the blow of the hammer. This phenomenon is known as myoidema, and it indicates an undue irritability of muscular tissue. It has no diagnostic significance other than its presence in cases in which the muscles are undergoing wasting.

The presence of paralysis indicates a lesion in the pathway traversed by motor impulses. This pathway extends through the brain, spinal cord, and peripheral nerves, the only exception to this statement being those subserving motor impulses along the course of cranial nerves.

The starting-point of motor impulses is found in the cerebral convolutions bordering on the fissure of Rolando, known as the cortical motor areas. The fibres originating here compose part of the white matter of the cerebrum. They converge in their downward course until they reach the internal capsule, at which place they become quite compact, and constitute the anterior two-thirds of the posterior limb of that structure. From the internal capsule they enter the corresponding crus cerebri, of which they form the middle two-fifths. They next enter the pons, after emerging from which they form the anterior pyramids of the medulla oblongata. At the lower end of the medulla there occurs an incomplete decussation of the motor fibres, the majority of the latter crossing to the opposite side and continuing down the spinal cord in the posterior portion of the lateral columns. We call this bundle of fibres the crossed pyramidal tracts. The fibres which do not decussate, continue down the spinal cord, occupying a position adjacent to the anterior median fissure, thus forming the direct pyramidal tracts or columns of Türck.

Both the crossed and direct pyramidal tracts become smaller as they travel down the spinal cord, "owing to the fact that many of their axones and collaterals are constantly bending forwards and inwards to enter the gray matter of the anterior horn of the same side, there terminating in arborizations about the motor-cells." From the latter new fibres are given off, and these form the anterior or motor-roots of the spinal nerves, and, uniting with the posterior nerve-roots, make up the various spinal nerves.

This short presentation of the anatomical facts demonstrates that in any case of paralysis the lesion must be

1. **Cerebral,** the interruption being within the cerebrum, peduncles, pons, or medulla.
2. **Spinal,** the lesion being in the pyramidal tracts or motor-cells of the anterior cornua.
3. **Peripheral,** the lesion being somewhere along the course of one or more of the peripheral nerves.

Cerebral paralyses are of the hemiplegic and monoplegic types, generally, however, of the former.

Spinal paralyses assume the paraplegic type.

Peripheral paralyses are characterized by loss of power in muscles supplied by individual nerves, and, excepting in case of multiple lesions, are monoplegic in type.

It is now customary to refer to all paralyses dependent upon lesions of the motor-tract within the cerebrum, peduncles, pons, medulla and pyramidal tracts, as due to disease of the upper or central motor neurons; those originating in the motor-cells of the anterior cornua, and the nerves passing from the same, as due to disease of the lower or peripheral motor neuron. This division is an eminently practical one, for paralyses dependent upon lesion of the central neurons are associated with spasticity to some extent, at least; while those originating in the peripheral neuron are characterized by flaccidity and atrophy of the functionless muscles.

Nevertheless, paralyses dependent upon intracranial lesions present well-defined features constituting them a distinct class, from a clinical standpoint. Accordingly, I shall treat of them apart from those dependent upon disease of the pyramidal tracts of the medulla and spinal cord.

Cerebral Paralyses.

The diagnosis in any case of paralysis involves the determination of the seat and the nature of the lesion. The former is based upon the principles of cerebral localization; the latter upon the history of the case and the principles of pathology. Cerebral localization is best studied in a separate chapter; and for the pathological diagnosis, the following schema, modified from Bastian, gives a good outline:

Cerebral Paralysis from Lesions of Sudden Onset.

I. **Traumatism.**
  a. *Lacerations of brain substance or compression by depressed bone.*
  b. *Hæmorrhages.*
    1. Meningeal.
    2. Intracerebral.
  c. *Inflammations.*
    1. Cerebritis.
    2. Meningitis.

II. **Hæmorrhages.**
  a. *Meningeal.*
    Usually following blows or falls, syphilitic vascular disease, arterial degeneration of chronic alcoholism, Bright's disease, and in children from violent paroxysms of coughing.
  b. *Intracerebral.*
    Resulting usually from miliary aneurysms and vascular degenerations from numerous causes, and occurring most frequently after middle life.

III. **Vascular Occlusions.**
  a. *Thrombosis.*
    1. Arterial.
      i. During convalescence from acute diseases or childbirth.
      ii. From endarteritis (syphilitic or otherwise) or arterial degenerations.
      iii. From feeble and irregular heart-action (in combination with either of the above causes).
    2. Venous.
      i. In longitudinal sinus. From general causes, altered blood states, plus a feebly acting heart.

      ii. In lateral sinus. Secondary to inflammation of the scalp and bone, and is either of traumatic origin or follows caries of the temporal bone.

b. *Embolism.*
  1. Single.
  2. Multiple.

Paralysis from Lesions of Slow Onset.

I. **Tumors.**
II. **Exostoses.**
III. **Pachymeningitis Chronica.**

I, II, and III, may occur with or without history of traumatism, or with or without evidence of syphilis, tuberculosis, or tumors in other portions of the body.

IV. **Abscess.**
  1. From traumatism.
  2. Suppurative middle-ear disease.
  3. Tuberculosis.
  4. Pyæmic states, secondary to infective lesions elsewhere.

Paralysis from Lesions of very Slow Onset.

I. **Disseminated and other Types of Sclerosis.**
II. **Meningo-Encephalitis.**
III. **Syphilitic, Alcoholic and Senile Degenerations.**

Before proceeding with a consideration of the data by means of which the various affections above tabulated may be differentiated, I shall present the clinical features characterizing paralysis from brain disease in general, and then study the special phenomena of particular types of cerebral palsies.

Hemiplegia.—As already stated, paralysis from brain disease assumes the type known as hemiplegia, *i.e.*, a paralysis of one lateral half of the body, the paralysis being on the side of the body opposite to that of the intracranial lesion. The hemiplegia may be complete, in which case the patient is unable to make any movement whatever of the affected parts; or it may be incomplete or partial, the vigor and amplitude of the movements being more or less impaired.

Complete Hemiplegia.—When the paralysis is complete, the patient is obliged to lie in bed utterly unable to move a single muscle of either arm or leg. There may or may not be associated paralysis of the face and tongue. The paralysis of the face may be on either the same or the opposite side as compared with the palsied limb. It is readily recognized by the drawing of the mouth to the healthy side, and the immobility of the muscles of the lower portion of the face on the affected side. The paralysis of the tongue is recognized when the patient attempts to protrude the tongue, that organ deviating, during the effort, towards the side of the tongue paralyzed. The lingual palsy may occupy the same relation to the hemiplegia as may the facial paralysis, *i.e.*, it may be on either the same or the opposite side as compared with the hemiplegia. The muscles of respiration move normally in ordinary action. The same is true of the muscles of mastication. If, however, the patient attempts to take a deep breath, thus bringing the extraordinary muscles of respiration into play, it will be observed that the chest on the affected side does not expand as freely as does that of the healthy side. The explanation of this phenomenon is found in an hypothesis first advanced by Broadbent. Certain muscles of the body functionate only in company with the corresponding muscles of the opposite side. Such muscles are believed to have bilateral representation in the brain. Hence, when one-half of the latter is diseased, the healthy side is capable of subserving the functions of the diseased side in this respect, and the functions of the bilaterally-acting muscles are not visibly impaired. The muscles of the lower extremities sometimes act in association with those of the opposite side, and they sometimes act individually. They may be said to have partial bilateral representation. In cases of cerebral palsy, therefore, the legs, while paralyzed, are not as markedly so as are the arms, because of this incomplete bilateral representation. Muscles which possess highly specialized functions, as those of the hands and fingers, are represented in but one-half of the brain, hence are most severely affected; and, moreover, their loss of power, when the lesion is at all well marked, is permanent.

When hemiplegia is of sudden onset, it is often attended by loss of consciousness. Under such circumstances, the loss of power can only be recognized when the paralysis is complete, as indeed it usually is in such cases. It will be observed that when the patient is restless he moves the limbs on one side of the body only; or manipulation of the parts will demonstrate marked flaccidity on the paralyzed side, while some resistance to passive movements is encountered on the well side. The superficial reflexes on the paralyzed side are usually abolished. When the patient is conscious and the hemiplegia incomplete, it will be discovered that the arm is more markedly paralyzed than the leg, and the peripheral parts of the limbs more helpless than those centrally situated. If the patient is able to walk, he presents the characteristic hemiplegic gait already mentioned.

Variations from the above-described typical hemiplegia are often observed, and depend upon the situation of the lesion and the rapidity with which it takes place.

We are now ready for the study of the various pathological conditions of the brain capable of originating hemiplegia.

Paralyses of Sudden Onset.—Suddenness of onset is surely indicative that the lesion must be either traumatic or circulatory in origin.

I. **Traumatism.**—As stated in the tables, cerebral traumatisms include lacerations of the brain structure, hæmorrhage, compression from depressed cranial fractures, and inflammations. I have taken no account of cerebral concussion in the above classification, because I look upon this so-called condition as a myth originated in ignorance and producing untold harm for those suffering from intracranial injuries. The idea usually conveyed by the term "cerebral concussion" is a condition characterized by loss of function without alteration of structure, and produced by injury. Such a condition is inconceivable. The majority of cases in which concussion is diagnosed are really examples of cerebral laceration or meningeal hæmorrhage. The harm done by those who hold to this antiquated pathological absurdity is that they neglect the proper treatment of lesions perfectly amenable to rational treatment, thereby causing the loss of many lives, and reducing many more to hopeless invalidism.

(*a*) **Laceration of Brain Substance and Compression by Depressed Bone.**—The symptoms in this class of cases come on immediately after the reception of the injury. As a rule, loss of consciousness is the most prominent. Paralysis does not take place unless the Rolandic area or the motor paths are affected. The diagnosis at best is uncertain. Cerebral lacerations may be produced even though the cranium remains intact, in which case a correct conclusion cannot be reached with any degree of certainty. The fact that a traumatism has produced a profound disturbance of cerebral functions is, however, strongly suggestive. The compression of the brain by depressed bone fragments may also escape recognition, if one depends upon palpation of the scalp alone for diagnosis. Many have been the serious fractures observed by my colleague, Dr. W. B. Van Lennep, and myself, in which the true condition of affairs was made evident only after exploratory incision of the scalp. So strongly do we feel on this subject that we do not hesitate to urge routine exploratory incisions of the scalp in all cases of severe head injuries with disturbed cerebral function.

(*b*) **Hæmorrhages.**—(i) **Meningeal Hæmorrhage.**—Meningeal hæmorrhages may be either sub- or extra-dural. The differentiation of these varieties is not possible during life. The one symptom which is surely diagnostic of this condition is a *latent period of undisturbed function between the time of the reception of the injury and the time of onset of the symptoms.* I would not hesitate to diagnose meningeal hæmorrhage on this one symptom, and yet it is far from uncommon to find such cases misnamed "concussion." When the effusion of blood is over the motor area, the localized palsy or convulsions add strength to the diagnosis; but when, as usually happens, the loss of consciousness is the only clinical manifestation, one can make a topographical diagnosis only by the discovery of an associated cranial fracture, or by his knowledge of the blood-vessels, injury of which is most likely to give rise to this condition.

(ii) **Intracerebral Hæmorrhage.**—Traumatic hæmorrhage into the brain substance cannot take place without laceration of structure of that organ. The symptoms of the two conditions must be identical, those of hæmorrhage being the more profound, because attended by the greater destruction of tissue.

(*c*) **Meningitis; Cerebritis.**—The traumatic inflammatory lesions manifest their symptoms only after the lapse of a few days after the reception of the injury. The time of onset constitutes a most important diagnostic factor. A differentiation of these two conditions, when of traumatic origin, is not possible during life. A further study of intracranial inflammations will be presented when treating of the non-traumatic varieties.

II. **Intracranial Hæmorrhage.**—(*a*) **Meningeal Hæmorrhage.**—The diagnosis of meningeal hæmorrhage is based upon both etiological and symptomatic factors. As already stated, injury is a most important cause. The distinctive feature of the traumatic cases is the interval of freedom from symptoms between the onset of the latter and the reception of the injury. This interval may be but a few minutes, when the escape of blood is rapid and profuse, or it may be of several hours, or even of a day's duration, the patient going about his ordinary work in the meantime, entirely unaware of his serious condition. While meningeal hæmorrhage may occur at any time of life, nevertheless an unusually large proportion of cases are observed in infancy, childhood, and early adult life. In children, scarlatina and its attendant nephritis, violent paroxysms of coughing, as in whooping-cough, and the vascular degenerations attendant upon the acute infections, constitute important causes. In adults, the lesion is favored by syphilis, alcoholism, the various forms of Bright's disease, etc.

The effusion of blood may take place between the dura and the cranium (extra-dural hæmorrhage), between the dura and pia-arachnoid (subdural hæmorrhage), or between the arachnoid and the cerebral cortex (subarachnoid hæmorrhage). In either of these varieties the symptoms are the same, as they depend upon the mechanical effect of the effused blood upon the brain as a whole, and upon disturbance of the functions of that particular portion of the cortex underlying the clot.

The symptoms related to general cerebral disturbance are identical in character with those observed in many other diseases of the brain, and are known as *diffuse symptoms*. Their manner of onset only is of diagnostic importance. The symptoms arising from the position of the clot are unquestionably of greater practical value. The fact that the symptoms exhibited by the patient in any case of intracranial hæmorrhage are limited to a disturbance of function of one or another part of the cerebral cortex, especially if their progress shows an extension of the lesion from one cortical area to its neighbors, argues strongly for the superficial situation of the hæmorrhage. Unless relief is obtained by surgical operation, the natural course of cases of meningeal hæmorrhage is progressive, coma finally becoming profound, and ending fatally.

The almost invariable source of the bleeding is the middle meningeal arteries and their branches. According to the one of the latter from which the bleeding proceeds, meningeal hæmorrhage has been divided into (i) *fronto-temporal*, (ii) *temporo-parietal*, and (iii) *parieto-occipital*. As a rule, the localizing symptoms will enable the physician to determine the seat of the injury.

There are two symptoms often present and possessing high diagnostic value. The first of these is a dilated and fixed pupil on the side corresponding to that of the lesion, and the other is conjugate deviation of the head and eyes to the side of the lesion.

The clinical course of a case of meningeal apoplexy presents at times important differences from that of intra-cerebral hæmorrhage. The moment of onset is marked by unconsciousness as the result of the attendant shock. From this the patient not infrequently recovers, only to relapse as the hæmorrhage progresses. This series of phenomena may be said to correspond with those observed in traumatic cases in which there is a period of freedom from symptoms following the reception of the injury.

Non-traumatic meningeal hæmorrhages offer a highly unfavorable prognosis, because they are almost invariably dependent upon a sadly degenerated condition of the blood-vessels.

Meningeal Hæmorrhage in Infants.—Meningeal hæmorrhage constitutes one of the etiological factors in the cerebral palsies of infancy and childhood. Important as it is, it must not be regarded as the only primary pathological change. As in adult life, cerebral paralysis may be due to embolism, thrombosis, inflammation and degeneration. Those originating during intra-uterine life may be dependent upon hæmorrhage produced by traumatism of the mother. Cases have also been reported in which the only assignable cause was maternal infectious disease of some kind. Cases originating at birth are nearly always dependent upon meningeal hæmorrhage. Those originating during infancy and childhood are due to the same causes as produce corresponding conditions in adult life, and their diagnosis rests upon the same principles.

(*b*) **Intracerebral Hæmorrhage; Apoplexy.**—Hæmorrhage into the brain substance is only possible when there is a degenerated condition of the cerebral blood-vessels. Hence a history of factors capable of producing vascular degeneration is an important guide in diagnosis. Among these causes may be mentioned syphilis; renal disease, especially interstitial nephritis; excessive alcoholic indulgence, extending over a term of years; various acute infectious diseases, notably typhoid fever, scarlatina and diphtheria; serious blood-changes, as purpura, scurvy, pernicious anæmia and leucocythæmia; laborious occupations and sedentary habits in association with high living, and the atheromatous changes of advancing years. Of these causes, syphilis is the most important in young adults; indeed, the incidence of apoplexy at that time of life is of itself strong presumptive evidence of a syphilitic vascular disease. In middle life and beyond, interstitial nephritis and atheroma are the important causes. The infectious fevers account for the majority of cases in childhood. The exciting causes include such as serve to bring about increased vascular pressure in the brain, including straining at stool, violent paroxysms of coughing, inordinate muscular exertion, and sudden revulsion of blood from the cutaneous surface, as from sudden exposure to a cold atmosphere.

The onset of an attack of apoplexy is always sudden, and may or may not be associated with unconsciousness. Any premonitory symptoms are dependent upon imperfect nutrition of the brain due to degeneration of the blood-vessels of that organ. They are always of an indefinite character, and include such as vertigo, mental and physical weakness, sensory disturbances, etc. The extent of initial unconsciousness varies in different cases all the way from the most profound coma to a simple temporary obscuration of intellect. When the stupor is profound, it is attended by stertorous breathing. The pulse is full and strong. The pupils usually do not react to light. Examination will determine the presence of paralysis of the arm and leg of one side of the body. Sometimes one side of the face is also paralyzed. In those cases of apoplexy in which consciousness is not disturbed, the recognition of the paralysis is an easy matter. At the commencement of an apoplectic attack the temperature is usually depressed, owing to the shock. In the course of a few hours it rises, ordinarily not higher than 103° F.; but instances are not uncommon in which it reaches 106° F., or more,—such cases offering a very unfavorable prognosis.

If the patient is to recover, consciousness is regained within a few days and the paralysis lessens, the muscles of the leg improving more rapidly than those of the arm. It is rare that the loss of power of the leg does not improve sufficiently to enable the patient to walk to some extent.

III. **Vascular Occlusions.**—(*a*) **Thrombosis.**—The causes leading to thrombosis of the cerebral arteries have already been tabulated. (*Vide* p. 701.) It will be seen that this lesion, consisting, as it does, of a coagulation of the blood within the obstructed artery, must be occasioned by alterations in the vascular walls, increased coagulability of the blood resulting from alterations in the composition of that tissue, or weakness of cardiac action, possibly in association with the two conditions first mentioned. A knowledge of the possibility of such arterial changes being present constitutes an important factor in making the diagnosis. Those favoring changes in the arterial walls include atheroma, syphilitic disease, gout, alcoholism, prostrating illness, and chronic Bright's disease. The causes originating in altered composition of the blood include pregnancy, acute infectious diseases, cancer, gout, tuberculosis, general malnutrition, and chlorosis. Cardiac weakness sufficient to excite the lesion under discussion may attend any of the diseases of the myocardium already mentioned in a preceding chapter. (*Vide* p. 493.)

Cerebral thrombosis must from its very nature be preceded by symptoms of general character resulting from the impoverished condition of the brain, or from general malnutrition. The attack itself is sudden, though never as rapid in its onset as in the case of hæmorrhage or embolism. In syphilitic cases there are often preceding headache and cranial nerve palsies. In those of atheromatous origin, the premonitory phenomena include vertigo, temporary aphasia, numbness of the extremities, drowsiness, and transient disturbances of speech.

In the majority of cases the onset of thrombosis is gradual, several hours being required for a full development of the symptoms. In others it may appear to be sudden, and it may even come on during sleep. Unconsciousness is not as likely to be an initial phenomenon as in hæmorrhage, the first manifestation being a gradually increasing hemiplegia. The febrile phenomena are the same as those observed in hæmorrhage, *i.e.*, initial fall with subsequent rise of temperature. Recovery of the paralysis is apt to be more rapid than in hæmorrhage. With the subsidence of the acute stage, the symptoms present no distinctive features to differentiate the case from one of cerebral hæmorrhage.

In case of recovery from the attack, the prognosis as regards subsequent seizures is bad, owing to the associated vascular degenerations, cardiac weakness, and blood changes.

The portions of the brain tributary to the occluded vessel undergo rapid softening unless the collateral circulation is soon established.

(*b*) **Embolism.**—Embolism of the cerebral arteries may be either single or multiple. The latter is so commonly secondary to other diseases, the phenomena of which are both serious and obtrusive, that it seldom calls for extended clinical study. It is different, however, with an attack of embolism due to obstruction of a single artery.

In embolism premonitory symptoms are absent, the tissues of the brain being normal up to the instant at which the obstruction takes place. As in the case of hæmorrhage and arterial thrombosis, the etiological factors of the case are important from a diagnostic standpoint. There must be present some cause capable of producing embolism ; and the diagnosis is further assisted if the conditions productive of thrombosis and hæmorrhage are absent. A diagnosis of embolism is therefore favored by the youth of the patient and a history of acute or chronic endocarditis, the infectious fevers, anæmia, and other blood changes.

The onset in embolism is always sudden, and may consist of unconsciousness, hemiplegia, or convulsions. Unconsciousness is, however, often absent; but, when present, is usually not of long duration. The patient does not present the flushed face or the high arterial tension of cerebral hæmorrhage, and the temperature changes already mentioned as characteristic of thrombosis and hæmorrhage are absent.

In the majority of cases of embolism the obstruction is in the left middle artery; hence right hemiplegia and aphasia are common symptoms of this lesion. The chronic stage of embolism does not differ from that of hæmorrhage.

Embolism is always to be regarded as a serious lesion. Its prognosis is, in the main, better than that of hæmorrhage, and there is not as great liability to recurrence.

As in thrombosis, there is softening of that portion of the brain to which the branches of the occluded artery are distributed.

It is often of clinical interest to determine the particular vessel occluded. Hence the following from Birch-Hirschfeld and Dana may be found useful:

"Softening in the region of the frontal convolutions on the inner surface of the hemispheres as far as the callosomarginal sulcus—due to obliteration of the trunk of the anterior cerebral artery.

"Total softening of the territory of the artery of the fissure of Sylvius, including the corpus striatum—due to obliteration of the first two centimetres of this artery.

"Total softening of the cortical areas supplied by the artery of the fissure of Sylvius, the corpus striatum excluded—due to occlusion of the artery just beyond the giving-off of the arteries of the corpus striatum.

"Partial softening in the area supplied by the arteries of the fissure of Sylvius—due to embolism of the inferior external frontal artery. The result is a softening of the island of Reil and of the third frontal convolution. If this is on the left side, aphasia results.

"Softening in the posterior part of the second frontal convolution and in the first central convolution—due to obliteration of the anterior parietal artery.

"Softening of both central convolutions and the Rolandic fissure, the anterior part of the first parietal convolution, and the island of Reil—due to closure of the median parietal artery.

"Softening of the lower parietal convolution and the first temporal convolution, as well as the island of Reil—due to obliteration of the posterior parietal artery.

"Softening in the area of the posterior cerebral artery rarely complete, involves the inferior part of the occipital lobe and the tip of the occipital lobe.

"Total softening of the corpus striatum, including the capsule, the lenticular and caudate nucleus, and the anterior third of the thalamus—seat of thrombus in the beginning of the artery of the fissure of Sylvius.

"Partial softening in the form of a cone whose apex lies in the anterior part of the lenticular nucleus, while the base, directed forward, involves the anterior two-thirds of the corpus striatum ; the cone is formed by the anterior part of the caudate nucleus, the internal capsule, and the third segment of the lenticular nucleus—the seat of occlusion lies in the lenticulo-striate artery.

"Partial softening lying more posteriorly than the preceding, involving the postero-external part of the lenticular nucleus, the internal capsule, the anterior part of the thalamus, and the tail of the corpus striatum—the lesion involves the lenticular optic artery.

"Partial softening of the thalamus opticus ; the lesion ranges in size from a pea to a hazel nut ; the vessels involved are the perforating arteries of the choroid plexus.

"Softening of the frontal, parietal and sphenoidal lobes—due to embolism at the point of bifurcation of the internal carotid, extension of the thrombus into the anterior cerebral artery."

Thrombosis of the Sinuses.—This may be of two kinds : (a) septic thrombosis, and (b) marantic thrombosis.

Septic Sinus Thrombosis.—In this disease the sinus thrombosis is dependent upon infection from some septic disorder in the territories drained by the veins emptying into the sinus affected. Thus, in the case of suppurative disease of the middle ear, the lateral sinus is involved. Taking, first, the symptoms of septic sinus thrombosis in general, it may be stated that they are those of constitutional sepsis, together with manifestations of a serious cerebral disorder. The general symptoms include rigors, fever of irregular type, showing remarkable excursions of the thermometer, and prostration. The cerebral symptoms include delirium, intense headache, vomiting, convulsions, double optic neuritis, nystagmus, strabismus, coma, and sometimes paralysis. A valuable aid in the diagnosis of the disease is afforded by the œdema localized over the area of distribution of the veins emptying into the affected sinus, and due to inflammation of the tributary veins. We now come to consider the special features of septic thrombosis of the individual sinuses.

Thrombosis of the Lateral Sinus.—This is practically always dependent upon chronic suppurative disease of the middle ear. An especially valuable diagnostic sign is œdema back of the ear, due to the extension of the thrombus to the small veins which pass through the mastoid and posterior condyloid foramina. There will be tenderness over the œdematous area. Gerhardt has shown that the internal jugular vein on the affected side is comparatively empty, a condition which is best demonstrated, clinically, during efforts at inspiration. It may be that the inflammation involves the internal jugular vein, leading to tenderness in this locality; or the thrombus may extend into the latter vein, discoverable as a firm, painful cord on palpation of that vessel.

Thrombosis of the Cavernous Sinus.—This is commonly due to infections from the eyes and the orbits. The special symptoms include exophthalmos on the side corresponding to the affected vessel, chemosis of the lids, contracted pupils, distention of the retinal veins, circumscribed areas of sweating about the forehead, and paralysis in the area supplied by the third cranial nerve; later, the fourth and fifth cranial nerves exhibit disordered function. In the beginning of the affection the symptoms are unilateral, but, later, may extend to the opposite side by reason of extension of the thrombus.

The diagnosis of septic sinus thrombosis is ofttimes made difficult by the association of septic leptomeningitis and cerebral abscess.

Marantic Thrombosis.—This variety has also been called primary thrombosis, and is a rare disease. It is especially liable to occur in exhausting diseases, such as diarrhœa and marasmus of infants, and in adults during the closing hours of certain diathetic conditions, as cancer and tuberculosis. The symptoms of these several affections are so obtrusive, as a rule, that it is not uncommon for them to obscure those of the sinus thrombosis, which is first discovered at the autopsy. The sinus usually affected is the superior longitudinal. The resulting symptoms are deducible from the anatomical conditions. Inasmuch as the superior longitudinal sinus has important relations with the veins of the nose, we find the mucous membrane of that organ highly congested, even to the extent of causing nose-bleed. There may also be marked fullness of, and even cyanosis in, the area of distribution of the anterior facial vein. Œdema of both temporal regions, spreading up to the vertex, occurs, especially in children, owing to obstructed circulation in the veins of the scalp which empty into this sinus by the way of the veins of Santorini. In children, also, it is not uncommon for the fontanelles to be protruding. The associated cerebral symptoms are not diagnostic, being such as may occur in conjunction with any serious and profound intracranial disorder, and include delirium, headache, vomiting, coma, convulsions, and paralysis.

IV. **Inflammations.**—(*a*) **Of the Brain.**—(1) **Acute Focal Encephalitis.**—This corresponds to the condition denominated by Strümpell acute polioencephalitis. The latter term is hardly correct, because the inflammation in these cases, though mainly involving the gray matter, is not limited to that structure. It is an affection occurring in infants and young children, for the most part, and is the primary lesion of many of the cases of spastic hemiplegia and diplegia of infancy and childhood. It seems to be especially liable to occur after or during the course of certain acute infectious diseases, notably typhoid fever, measles, influenza, diphtheria, scarlatina, and variola. It may also result from syphilis, alcoholism, and traumatism.

Attacking children, acute focal encephalitis sets in with fever, vomiting, and convulsions. It may be that the acute manifestations of the disorder are preceded by certain symptoms of indefinite significance, as headache, irritability, vertigo, and drowsiness for several days. The convulsions may be general or local, usually the latter, and are followed by paralysis of hemiplegic or diplegic type. Following these symptoms, the patient may become comatose or delirious. In severe cases the respiration assumes the type known as Cheyne-Stokes, or it may be greatly embarrassed. Sometimes the inflammation becomes diffuse, as shown by symptoms referable to disturbance of the cranial nerves. The case may end fatally within one or two weeks. If the patient recovers, there nearly always remain lasting deformities, including hemiplegia or diplegia, with contractures, epileptic seizures, imbecility, athetoid movements, etc.

In adults, acute focal encephalitis produces phenomena very similar to those observed in the case of children. The subsequent paralyses with contractures and the epileptiform convulsions are not apt to be so severe or extensive.

The recognition of acute focal encephalitis is not easy. It is very liable to be mistaken for hæmorrhage, embolism, and meningitis. Hæmorrhage is not likely to be preceded by the initial or premonitory symptoms; and the same is true of embolism. The fever in these affections, moreover, is not as early a symptom as it is in encephalitis. In embolism, also, we get a history of endocarditis and rheumatism.

(2) **Polioencephalitis Superior.—(Nuclear Ophthalmoplegia.)**—The lesion in this disease is an acute hæmorrhagic inflammation involving the cerebral gray matter in the floor of the third ventricle and the aqueduct of Sylvius. It is a rare disease, and is believed to be due to alcoholic excesses and the acute infectious processes. The onset of the disease is sudden, although it may be preceded by paræsthesiæ of the extremities, vertigo, diplopia, and headache. The symptoms of the disease itself closely resemble the phenomena of delirium tremens. In the course of three or four days the ophthalmoplegia becomes manifest, first as ptosis, and later as immobility of the eyeballs, the ciliary muscle remaining unaffected. There may also be double optic neuritis, incoördination of gait, diminished or absent reflexes, and disturbances of speech. Usually the muscles of the face escape. The patient is at first delirious, later becoming comatose. Cases presenting the latter symptom rarely recover.

Sometimes disturbances of gait precede or are associated with the ophthalmoplegia. These consist for the most part of incoördination or ataxia. Sometimes they are so prominent on one side of the body as to suggest a hemiplegia.

(3) **Acute Hæmorrhagic Encephalitis.**—This affection is due, as a rule, to infectious processes,—notably those of influenza, typhoid fever, typhus, and the puerperal state. Its manifestations are of sudden onset, and include severe headache and high fever, soon followed by vertigo, vomiting, photophobia, and delirium. Next the patient goes into a condition of stupor, from which he can, as a rule, be partially aroused. The respirations are shallow and frequent, and the pulse rapid and feeble; the reflexes become enfeebled, and the sphincters incompetent. Accidental symptoms include epileptiform convulsions, aphasia, paralyses of various types, hemianopsia, hemiataxia, nystagmus, and difficulty in speech and deglutition. In its milder forms it runs a course of two or three weeks, after which the patient gradually recovers. It is a dangerous disease, and in fatal cases the coma deepens, and the patient dies of exhaustion. It differs from meningitis in that the coma appears early, and the pupils do not present the marked contraction observed in that disease, and the stiffness of the neck, the rigidity of the limbs and the general hyperæsthesia are absent. Etiological factors, *e.g.*, grippe or other infectious diseases, are important in diagnosis.

Paralyses from Lesions of Slow Onset.—I. **Tumors.**—The etiology of tumors being for the most part enshrouded in doubt, causative factors afford but little aid in diagnosis, unless we except those cases in which there is a plain history of syphilis or tuberculosis. The symptoms suggesting the diagnosis of tumor include two classes: (a) Those of a diffuse character, and (b) those indicative of a localized lesion. The diffuse symptoms of cerebral tumor include headache, vomiting, optic neuritis, vertigo, and mental disturbance. Of these, headache is probably the most constant, being present in practically all cases. Of course notable exceptions to this statement will be observed. It partakes of the usual characteristics of organic headache, being always with the patient, though subject to periods of exacerbation. Its location does not bear any definite relation to the situation of the tumor, excepting when the latter involves the meninges or is superficially situated in the cerebral substance. Vomiting is much less frequently observed than is headache. It exhibits the usual projectile character of cerebral vomiting. It is especially liable to be present when the tumor involves the cerebellum or the structures about the base of the brain. Double optic neuritis constitutes the most important symptoms of intracranial neoplasm. In fact, its occurrence in association with the headache and vomiting above described is all but conclusive evidence of the presence of cerebral tumor. It is found in 80 per cent. of all cases, and is especially constant in tumor at the base of the brain. In tumors of the convexity it is so frequently absent that the negative evidence thus afforded counts for but little. Vertigo is present in about half the cases, especially in those involving the cerebellum. Impairment of the mental faculties is not characteristic of cerebral tumor, excepting in those cases in which the growth involves the frontal lobes. Nevertheless they may occur in conjunction with tumors elsewhere, when the size of the growth is such as to interfere with the functions of that portion of the cerebrum. Generalized epileptiform convulsions are not infrequent. The focal symptoms include phenomena of irritation and depression. Under the former may be included convulsive seizures of Jacksonian type, and various manifestations referred to the centres governing the special senses. Examples of depression symptoms (which must result from destructive lesions) include paralyses dependent upon destruction or pressure upon motor and sensory centres and tracts. Thus we have present hemiplegia, paralyses of the cranial nerves, hemianæsthesia, aphasia, hemianopsia, etc. To make these symptoms of value diagnostically, they must follow the clinical course of those of cerebral tumor, *i.e.*, they must be slowly progressive. Nevertheless, frequent exceptions to this may be observed. Thus the tumor may exist for a long time without the slightest impairment of health, when, without apparent reason, symptoms suddenly appear, and death closes the scene. The conditions which may be confounded with brain tumor include hysteria, uræmia, lead-poisoning, and abscess.

The diagnosis of the nature of the tumor can never be made with any certainty. Still it should be attempted. Tubercular tumors are especially liable to occur in childhood and early life. They are apt to be associated with tubercular disease in other portions of the body, systemic poor health, and a tubercular family history. A syphilitic growth is to be suspected especially in males and when a history of syphilis is obtainable, when the patient is a young adult, and there is considerable meningeal irritation. Gliomata are characterized by sudden exacerbations of symptoms due to hæmorrhage within their structure; irritative phenomena are prominent; and the family history of tuberculosis and a pre-existence of syphilis can be negatived. Carcinoma is suggested by the age of the patient, *i.e.*, past middle life, and a family history of malignancy.

II. **Abscesses of the Brain.**—In the diagnosis of cerebral abscess the etiology of the illness plays a necessary part. Unless one of the well-known causes of this condition is present, it is impossible that the lesion be abscess. These causes include traumatism, producing an infected wound, chronic middle-ear suppuration, tuberculosis, and metastasis. As to the *rôle* played by traumatism in the production of brain abscess, there is great general misconception. It is not necessary that the injury be severe, that the cranium be fractured, or the cerebral substance lacerated—the one essential factor is an infected wound of the soft parts of the head. The injuries most liable to produce cerebral abscess are punctured wounds, penetrating to or into the bone, and punctured fractures of the skull; compound fractures, with large open wounds, are not at all liable to be thus complicated, as their serious nature is at once suspected and efficient treatment is instituted early. Pathogenic infections of the face and scalp, *e.g.*, erysipelas, may produce abscess, but are far more likely to generate thrombosis or septic meningitis. Chronic middle-ear suppuration is probably responsible for one-half of the cases of cerebral abscess. When acute suppurative otitis media produces brain abscess, infection is carried into that organ by the lymphatics, evidence favoring this view being found in the presence of enlarged cervical and post-auricular glands. Chronic nasal disease must not be forgotten as an occasional cause of brain abscess, as it is also of septic meningitis. The number of cases resulting from tuberculosis and metastasis is relatively small. The latter class is observed in conjunction with septic endocarditis, and is due to infectious emboli.

The symptoms of cerebral abscess resemble those of tumor in great measure. There are, of course, the usual phenomena suggestive of suppuration. Thus, in the acute cases there are not infrequently rigors and high temperature. In the chronic cases, however, the special clinical features of a suppurative process are absent. Indeed, rigors and fever are then suggestive of thrombosis or septic leptomeningitis rather than of abscess. Headache is commonly present, but is not as severe as in tumor. Projectile vomiting is common. Optic neuritis is by no means as frequently observed as in tumor. Focal symptoms are not as clearly defined as in tumor. One is usually obliged to rest his localization of the lesion upon etiological factors. Thus, when the abscess has resulted from middle-ear suppuration, the lesion is usually situated in the corresponding temporo-sphenoidal lobe or in the cerebellum. As to which of these localities is involved in any particular case, one is obliged to determine by the weight of the clinical evidence.

Spinal Paralyses.

The spinal paralyses may be divided, clinically, into classes, according to their rapidity of onset, as follows:

I. **Onset abrupt.**
  a. Fractures and dislocations of the vertebræ.
  b. Wounds of the spinal cord.
  c. Meningeal hæmorrhage.
  d. Intra-medullary hæmorrhage.
  e. Ischæmia of the lumbar enlargement of the cord.

II. **Onset acute.**
  a. Thrombotic softening.
  b. Acute myelitis.
  c. Acute poliomyelitis anterior.

- d. Caisson disease.
- e. Acute ascending paralysis.

III. **Onset subacute.**

- a. Intermittent paraplegia.
- b. Hysterical paraplegia.

IV. **Onset subacute or chronic.**

Subacute and chronic poliomyelitis anterior.

V. **Onset chronic.**

- a. Cancer of the vertebræ.
- b. Pachymeningitis from vertebral caries.

VI. **Onset very chronic.**

- a. Spastic paraplegia.
- b. Ataxic paraplegia.
- c. Amyotrophic lateral sclerosis.
- d. Locomotor ataxia.
- e. Disseminated sclerosis.
- f. Diffuse sclerosis (Friedreich's ataxia).
- g. Cervical hypertrophic meningitis.
- h. Tumors of the cord and meninges.
- i. Syringomyelia.

A classification based on the mode of onset is of special value as offering some suggestions as to the nature of the pathological changes. We may also study another classification based upon the associated symptoms. This offers suggestions as to the portions of the spinal cord affected, and, taken in conjunction with the mode of onset, is of great diagnostic value.

I. **Spinal paralyses associated with muscular atrophy.** This class of cases is usually unattended by pain or disorders of sensibility. If the latter are present, they are not prominent features of the case.

(1) ACUTE:

Poliomyelitis anterior.

(2) CHRONIC:

- a. Chronic poliomyelitis.
- b. Progressive muscular atrophy.
- c. Amyotrophic lateral sclerosis.
- d. Acute descending paralysis (Landry's paralysis).

II. **Spinal paralyses associated with severe pains in either the trunk or extremities.**

- a. Locomotor ataxia.
- b. Cervical hypertrophic meningitis.
- c. Malignant disease of the vertebræ.
- d. Tumors of the cord and meninges.
- e. Pachymeningitis with vertebral caries.

III. **Spinal paralyses not associated with early muscular atrophy or severe pains.**
  a. Ataxic paraplegia.
  b. Spastic paraplegia.
  c. Disseminated sclerosis.
  d. Diffuse sclerosis.
  e. Acute myelitis.
  f. Thrombotic softening.

Spinal Paralyses of Abrupt Onset.—As seen by reference to the tabulation according to mode of onset, the paralyses presenting abrupt modes of onset are those arising from traumatism and circulatory lesions.

The traumatic cases are to be recognized by the nature of the accident and the local objective conditions. Thus we may determine fractures and dislocations of the vertebra and wounds of the spinal cord. The extent of spinal-cord lesion is to be gauged by a clinical study of the functional condition of that organ, and a determination thereby of the special structures which have been damaged.

Meningeal hæmorrhage is nearly always traumatic in origin. Its recognition depends upon an interval of freedom from symptoms between the time of the reception of the injury and the appearance of the paralysis.

Ischæmia of the lumbar enlargement of the spinal cord is a very rare accident, and must arise from obstruction of the abdominal aorta. In such cases the lower extremities must be cold and pulseless.

I. **Differentiation of the Spinal Paralyses Associated with Muscular Atrophy, and Without Disturbance of Sensibility.**—Of the acute affections of the spinal cord belonging to this class, there is but one, namely, (1) **acute poliomyelitis anterior,** also known as *acute spinal paralysis of infants*, *atrophic paralysis*, *regressive paralysis*, and *essential paralysis of infants*.

This affection may occur at any time of life, but is far more common in infants and young children. Its etiology is in a great measure obscure. There is a growing belief that it is due to infectious causes, several epidemics having been reported in this country and abroad. It certainly does not appear to have any definite relation to dentition, indigestion, or other common disorders of early life, and it seems equally certain that it rarely follows upon traumatism. The vast majority of cases have their inception during the hot months of the year.

The initial symptom is usually fever, which may be of high degree, and even associated with delirium and convulsions, in exceptional instances. The paralysis comes on rapidly, after the fever has continued for a day or two. It may invade one or all four extremities. The paralyzed parts are flaccid. The deep and superficial reflexes are destroyed. The sphincters of the bladder and rectum are unimpaired, and sensation is retained. At a period ranging from four days to two weeks, according to the severity of the case, the paralyzed muscles manifest the reaction of degeneration. Beginning with the end of the first week, atrophy of the paralyzed muscles appears, and proceeds to a greater or less degree. Still later the skin gives evidence of malnutrition, becoming cold and clammy, and presenting a bluish or mottled appearance. Their surface-temperature may be ten or more degrees below the normal. If the case is a mild one, some improvement is noted at about the end of the first week. If the case is severe, it may be delayed for several weeks. At the end of three weeks most of the possible improvement may have been obtained. It is the rule for some muscles—how many, will depend upon the severity of the case—to remain permanently impaired. A very important and serious result of acute poliomyelitis is the deformities arising from defective growth of the bones and contractures of the muscles.

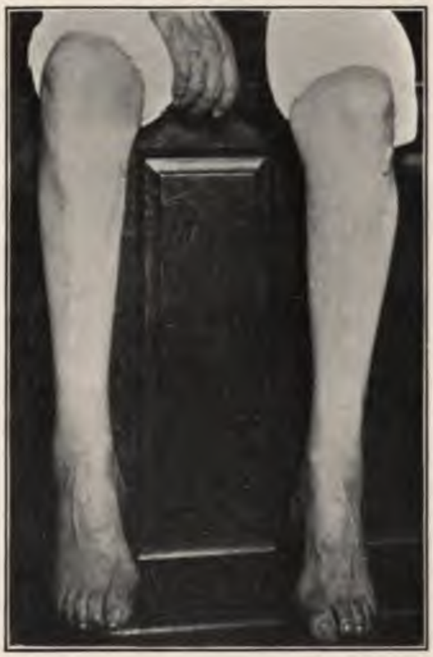
The acute poliomyelitis of adults differs from that of infants and children in the comparative mildness of the initial symptoms. The paralytic phenomena resemble those observed in infancy in every way. The parts having obtained their growth, there is little likelihood of resulting deformities.

(2) **Chronic Spinal Paralyses with Muscular Atrophy.**—(*a*) **Chronic Poliomyelitis Anterior.**—The paralysis is usually manifested first in the legs, and increases gradually. The arms are often implicated at the same time. The affected muscles are flaccid. The superficial and deep reflexes in the affected areas are lost. The paralyzed muscles undergo atrophy. The reaction of degeneration is present, and there is no involvement of the sphincters. Fibrillary twitching is almost always present. The progress of the disease varies greatly in different cases. In some the disease remains stationary after several months; in still others it decreases, a fair degree of improvement resulting. In still others it progresses to a fatal termination.

(*b*) **Spinal Progressive Muscular Atrophy.**—The spinal form of progressive muscular atrophy usually begins in middle life. Its etiology is obscure, although traumatism, exposure and heredity have been assigned as causes. The majority of cases occur in males. The first manifestation of the disease appears in the upper extremities as atrophy of the small muscles of the hands. The muscular wasting is noticed especially in the thenar and hypothenar eminences. The atrophy of the interossei leads to deepening of the interosseous spaces. Fibrillary twitching is always present. The electrical examination of the affected muscles elicits weak response in direct proportion to the extent of loss of muscular tissue. The paralytic phenomena present also go hand in hand with the degree of atrophy, and are directly dependent upon it. The natural course of the disease is progressive, one set of muscles after another being invaded. It may be interrupted by long periods of remission. The lower limbs do not participate in the symptoms until the disease is well-advanced. The prognosis of the spinal form of progressive muscular atrophy is in the highest degree unfavorable. It will readily be seen that this disease bears a close resemblance to chronic poliomyelitis. It differs from the latter, however, in that the atrophy and not the paralysis is the initial phenomenon, the absence of the reaction of degeneration, and in pursuing a slower and more certainly progressive course.

(*c*) **Amyotrophic Lateral Sclerosis.**—This affection depends pathologically upon a combined degeneration of the anterior gray horns and of the lateral columns. With the lesions in these situations, the symptoms can readily be surmised. According as the pyramidal tracts or the anterior horns are relatively more or less affected, so will spastic paralysis or muscular atrophy predominate. The first symptoms usually consist of numbness or formication in the fingers. Then follows motor weakness, with more or less rapidly progressive muscular atrophy. The atrophy affects the muscles as a whole, and not fibre by fibre, as in progressive muscular atrophy. Fibrillary twitching is commonly present. Soon muscular rigidity is noted, and this increases in intensity until it may amount to absolute contracture. When the spastic stage is fully developed, the arms

FIG. 191.



Atrophy of legs in a case of progressive muscular atrophy.

become firmly drawn to the body, the forearms bent on the arms, and the fingers strongly flexed. The legs become involved in the second stage of the disease. Here paraplegia with rigidity is the prominent symptom. The terminal stage of the disorder is usually characterized by involvement of the muscles of the lips, tongue, pharynx and larynx, and the mind is affected. Throughout the course of the disease the deep reflexes are greatly exaggerated. When the atrophy advances to a marked degree, it is usual for the spastic phenomena to decrease. The prognosis of this affection is highly unfavorable, the disease being surely progressive. Departures from the typical course as above outlined are not infrequent, and have been described on page 660.

(*d*) **Acute Ascending Paralysis; Landry's Paralysis.**—The propriety of considering acute ascending paralysis among the spinal palsies is open to question. Inasmuch as spinal changes consisting of inflammatory and vascular conditions or alterations in the nerve-cells have been regularly found by the newer methods of investigations, and its clinical study is convenient with the affections above described, I have decided to study it at this time. The disease is unquestionably dependent upon infectious causes and toxins, which in most cases are bacterial products.

The disease is ushered in by vague sensory phenomena, as aching and soreness of the parts about to be paralyzed. Then the patient notices a weakness in both lower extremities, and this increases rapidly even in a few hours, until the loss of power is absolute. The muscles first attacked are those of the feet; next in order come those of the legs; and, finally, those of the thighs. The lower extremities having been rendered functionless, the muscles of the trunk are next attacked and rendered helpless; and then those of the arms are treated in a like manner. The disease finally attacks the muscles of respiration and carries off the patient. The paralyzed muscles are flaccid, but rarely undergo any atrophy whatever. The electrical reactions are usually normal; exceptionally, reaction of degeneration may be observed. The deep and superficial reflexes are either deficient or destroyed. The bladder and rectum retain their normal functions. The prognosis of acute ascending paralysis is highly unfavorable, as it terminates fatally, as a rule, within a couple of weeks.

II. **Spinal Paralyses Associated with Severe Pains in Either the Trunk or Extremities.**—(*a*) **Locomotor Ataxia; Tabes Dorsalis; Posterior Spinal Sclerosis.**—This is, in all probability, the most frequently observed of all chronic affections of the spinal cord. A syphilitic history is so regularly obtainable in my experience that I have come to regard syphilis a necessary prerequisite to its existence. Other agencies may be operative as exciting or determining causes; but syphilis is the one great predisposing factor. The first symptom observed is usually the lightning or fulgurant pains. Even at this early stage the knee-jerks are absent. These two symptoms may continue for years, even twenty, before any disturbance of gait is manifested.

This disorder in gait is known as the ataxic gait, and has been described elsewhere. (*Vide* page 692.) It is especially manifested when the patient attempts to walk with his eyes closed, or in the dark. If requested to stand with eyes closed and heels and toes approximated, he staggers and falls. As the disease progresses, walking and standing become more and more difficult, until finally they are impossible. Eventually the upper portion of the spinal cord is involved. At first the patient notices a difficulty in reaching for or grasping an object unless his gaze is fixed upon it; or he may observe that certain delicate movements, which he could formerly perform with ease, become awkward. Anæsthesia of various portions of the body is a prominent feature. As a rule, it is not noticed by the patient until the disease has existed for a number of years. Careful examination in the early stages, when the only other symptoms present are the lightning pains and the absent knee-jerks, will discover patches and bands of anæsthesia about the trunk. The anæsthesia of the extremities adds to the difficulties of walking. The patient complains of a sensation as if the soles of the feet were padded, or as if he was walking on thick velvet carpet. Pain in the back or spinal tenderness is never complained of in locomotor ataxia. Their presence indicates meningeal involvement. Eye symptoms present themselves in great variety in locomotor ataxia. Of these, the Argyll-Robertson pupil is especially characteristic. This makes its appearance, as a rule, early in the disease. Myosis and mydriasis are likewise not uncommonly observed; or, again, there may be inequality of the pupils. Atrophy of the optic nerve is observed in a small percentage of ataxic patients. It is especially distressing in that it progresses to complete blindness. For some reason thus far not explained, the ataxic symptoms do not progress in these cases. Paralyses of eye-muscles are exceedingly common symptoms. In many instances they are not sufficiently marked to manifest themselves as squint, but produce diplopia. Paralytic ptosis is also an occasional symptom. These ocular palsies are apt to be but temporary. They may, however, appear and disappear a dozen times during the progress of the disease. Bladder and rectal symptoms are very common in ataxia. Urinary incontinence is an after-accompaniment of the late stages of the disease; in other cases the patient is unable to empty his bladder completely by reason of deficient expulsive power. The residual urine is apt sooner or later to set up cystitis. The rectal symptoms are not apt to be so prominent. They are usually manifested as deficient expulsive power. Sexual desire in the early stages of the disease is usually unimpaired. In the later stages it vanishes, erectile power being entirely lost. The so-called visceral crises constitute an important but infrequently recognized manifestation of ataxia. Those most frequently observed are the gastric and nephritic crises. In the former the pains are paroxysmal, of a burning or lancinating character, and are described by the patient as if a red-hot fork had been plunged through the epigastrium into the stomach. They are frequently associated with retching and vomiting. In some cases the vomiting exists without the pain, and may be so severe that the introduction of the smallest quantity of water into the stomach is sufficient to renew it. These crises usually last three or four days, and recur at intervals of a month or more. They may be associated with irregularity of the heart's action. Renal crises simulate renal colic from the passage of a stone along the ureters. Lastly, we have certain trophic symptoms, including the joint-lesions, the perforating ulcer, falling out of the teeth and nails.

(*b*) **Cervical Hypertrophic Pachymeningitis.**—Opinions seem to differ as to what constitutes this disease. Some claim that the hypertrophic inflammation of the spinal dura is always secondary to hæmorrhagic extravasations, and others describe a special affection in which pachymeningitis with inflammatory proliferation leads to marked thickening, even to ten times the normal. The alleged causes of the affection are exposure, over-exertion, traumatism, syphilis, and alcoholism. In the initial stage the disease is characterized by severe pains with disturbed sensation, due to compression of the posterior spinal nerve-roots. These pains involve the neck, occiput and arms, and are of the most agonizing character. After a period ranging from two to six months, the anterior nerve-roots become involved, and then there is paralysis with atrophy of muscles, especially of those supplied by the ulnar and radial nerves. The paralysis of the flexor muscles predominates, and this leads to a characteristic deformity, in which the hands are hyperextended at the wrists, the first phalanges are extended and the second and third are strongly flexed. Ultimately, the movements of the entire upper extremities become involved. In the terminal stage of the disease, the lower extremities become affected with a spastic paraplegia without muscular atrophy.

Some authors describe cervical hypertrophic pachymeningitis as a variety of *syringomyelia*. In this they are probably mistaken, because some cases of the latter affection present the clinical features of the disease under consideration. Syringomyelia usually begins in the cervical portion of the spinal cord, and may be associated with severe pains and muscular atrophies. The dissociation of sensory phenomena is strongly suggestive of syringomyelia.

(*c*) **Malignant Disease of the Vertebræ.**—The important malignant growths of the vertebræ include carcinoma and sarcoma. The former may appear as a primary lesion, but is usually secondary to malignancy elsewhere, especially of the mammæ, and occasionally of the stomach and uterus. In most instances the bodies of the vertebræ are first involved. The symptomatology may be inferred from the natural course of such lesions.

The early clinical phenomena relate to the vertebræ. There is severe localized pain with tenderness, aggravated by any motion or jarring. Quite naturally, the spine is held rigid. An objective examination of the parts does not disclose the presence of a tumor, unless it has involved the vertebral arches. The cancerous infiltration of the bony structures may develop deformity, or, what is said to be suggestive of malignant vertebral disease, actual shortening of the spinal column. The next set of symptoms relate to irritation of the spinal nerve-roots. The most important of these are the pains produced by irritation of the posterior roots; they are usually persistent, and become progressively severe as the disease increases, and, what is especially characteristic, *are practically always bilateral from the first.* Nevertheless, the limitation of the pain to one side should not lead us to negative a diagnosis of vertebral cancer. The symptoms of motor irritation are rarely as prominent as those of sensory character, and consist of spasms and contractures. Next come the symptoms produced by nerve-compression, and these include paralyses with atrophy and anæsthesia. Lastly come the symptoms which indicate involvement of the cord itself. These include phenomena indicating compression of the cord at the level of the lesion, and interference with the motor and sensory conducting tracts below the part compressed.

(*d*) **Tumors of the Cord and Meninges.**—In a general way it may be stated that the symptoms of spinal-cord tumor are those of a focal lesion, which, in its early stages, produces phenomena indicating central irritation, and subsequently others showing compression and destruction of a transverse segment of the cord and its nerve-roots. Of the earlier irritative indications, pain is prominent. It is characteristically bilateral, and is apt to appear the earlier and be more severe as the tumor involves or approaches the meninges. Girdle sensations, anæsthesia and hyperæsthesia are also prominent symptoms. The paralysis almost invariably begins in one lower extremity. The paralyzed muscles supplied by the nerve-roots given off from the affected segment of the cord undergo atrophy, and exhibit the reaction of degeneration. The reflexes from this segment of the cord are abolished, while those derived from below are exaggerated. The clinical course of intra-spinal tumor is sometimes characterized by attacks of myelitis or meningitis, or both.

(*e*) **Pachymeningitis with Vertebral Caries.**—In the diagnosis of this affection it is important to recognize the fact that the spinal symptoms are not the result of the deformity of the vertebral column, for they are especially apt to appear early in the course of the disease, before there is much breaking down of the bony structures. The intraspinal changes which disturb the function of the cord include a more or less extensive inflammatory growth which compresses the anterior surface of the spinal cord, arrest of the circulation with resulting œdema, and pressure from the displaced bones. The symptoms upon which the diagnosis of the condition is based consist of those dependent upon the vertebral caries, plus those due to irritation of the spinal nerve-roots and involvement of the spinal cord. The bone symptoms come first. They include, first of all, pain in the back, which, if not accurately localized by the patient, may be elicited by side-pressure on the spinous processes with the finger-tips. Pressure with a sponge soaked in hot water may also be used for the same purpose. The pain is persistent; is aggravated by any motion. There is more or less rigidity of the spinal column at or about the seat of pain. The patient endeavors to make himself as comfortable as possible by assuming such postures as will relieve the diseased vertebræ of their superincumbent weight. When the deformity —kyphosis—occurs, then, of course, the diagnosis is beyond question. The symptoms dependent upon irritation of the nerve-roots may appear during any stage of the disease. They include peripheral pains, which, being referred to different locations, may give rise to diagnoses which fall wide of the mark. I have known them to be attributed to rheumatism, gastritis, appendicitis, and gall-stones. Sometimes they are agonizingly severe, though usually less so than in cancerous disease of the vertebra. They may be accompanied by herpes zoster. Probably the first evidence of involvement of the spinal cord itself is found in the increased reflexes. The various superficial and deep reflexes originating in the spinal segments below the lesion exhibit all grades of exaggeration, from the mildest to the most extreme. Ankle-clonus is marked. The paraplegia is, in the majority of cases, of gradual onset. In some few cases it may reach its maximum intensity in two or three weeks from its commencement; and in still more exceptional instances it has been known to come on suddenly, in response to a slight traumatism, without apparent cause, and reach its acme over night. It is always of the spastic variety. In extreme cases the bladder and rectum may be paralyzed. When compression of the cord at the level of the lesion is at all great, paralysis and atrophy of the muscles supplied by the particular spinal segment thus affected will take place. From a prognostic standpoint, it is well to know that in no disease of the spinal cord is recovery more liable to ensue, despite the gravity of the symptoms, than in the one under discussion.

III. **Spinal Paralyses not Associated with Early Muscular Atrophy or Severe Pains.**—(*a*) **Ataxic Paraplegia.**—This is a disease of uncertain etiology, dependent upon a combined sclerosis of the posterior and lateral columns of the spinal cord. Its onset is, as a rule, very gradual. Its first symptom is a slight weakness or tired feeling in the legs on any exertion, and this is associated with unsteadiness or incoordination of gait. The patient finds himself unstable when attempting to stand with his eyes closed and heels and toes approximated. These symptoms increase gradually over a term of years until complete paralysis develops. With the increasing loss of power the incoördination becomes a less prominent feature, and rigidity of the paralyzed limbs appears. The lightning pains of tabes are absent. Pain in the back is of common occurrence. Occasionally the patient complains of dull pains in the legs, especially on fatigue. All the deep and superficial reflexes are greatly exaggerated; ankle-clonus is marked. The functions of the rectal and vesical sphincters and the sexual organs are often impaired to a marked degree. Sometimes mental impairment is observed. The prognosis of the disease is highly unfavorable.

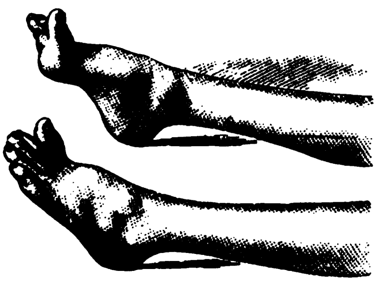
(*b*) **Spastic Paraplegia.**—This disease is likewise of uncertain etiology. It is dependent upon degeneration of the pyramidal tracts of the spinal cord. In a general way the symptoms consist of paralysis with rigidity of the affected parts, and a most remarkable exaggeration of the deep and superficial reflexes. The disease is of slow onset always. There is first noticed a slight weakness of the lower extremities, which gradually progresses to disability. At the same time the knee-jerks are found to be greatly exaggerated, and ankle-clonus is obtainable. The Babinski reflex is present. Rigidity appears, and is especially prominent in the extensor muscles. The spastic gait is characteristic. The limbs are moved as if in one piece. The toes scrape the ground. Sometimes, because of the strong adductor spasm, the limbs are drawn in front of each other, giving rise to the phenomenon known as cross-legged progression. The tension put on the tendo-Achilles excites an ankle-clonus, the tremulous movements of which are imparted to the gait. Sensory disturbances are absent, as a rule; if they do occur, they consist solely of sensations of numbness and formication. The sphincters are sometimes paralyzed. The arms will be affected if the degeneration involves the cord above the cervical enlargement. The prognosis is highly unfavorable.

(*c*) **Disseminated Sclerosis.**—Disseminated foci of sclerosis may invade the spinal cord and produce thereby a variety of symptoms limited only by the location and extent of the lesions. In this way may result clinical phenomena which may cause the disease to simulate any or all of the well-known system diseases of the cord,—which ones being dependent upon the particular portions of the spinal cord bearing the brunt of the pathological changes. It is evident, therefore, that no practical description can be given of disseminated sclerosis of the cord.

(*d*) **Friedreich's Ataxia.**—This disease is also known as hereditary ataxia. Strictly speaking, it is not an hereditary but a family disease. Its first manifestatious usually appear in childhood, although its advent may be delayed as late as the twenty-fifth year. The anatomical changes include a diffuse sclerosis of the posterior and lateral columns of the spinal cord.

Friedreich's ataxia is always of gradual onset. In many instances it may be impossible to determine the time at which the first symptoms appeared, because the weakness of the lower extremities appeared before the age at which the child should learn to walk. Under such circumstances, the difficulty is not unnaturally attributed to ordinary tardy development. In those who are already walking, the first symptom is an incoördination of gait, which resembles more the gait of alcoholic intoxication than of locomotor ataxia. It may or may not be aggravated when the eyes are closed. Next the arms are affected, and usually within a short time, as compared with the progress noted in other degenerative spinal-cord affections. Voluntary movements of the arms become irregular and jerky. Nearly always the knee-jerks are destroyed early in the disease. Very exceptionally, and then only in atypical cases of the disease, they are exaggerated. The further progress of the disease is characterized by involvement of the movements of the head and neck. These become irregular and jerky, and there may even be an irregular tremor. Speech becomes affected; syllables are omitted, and utterance is slow and hesitating. Nystagmus, observed especially when the patient is looking to one side, is nearly always present. Optic-nerve atrophy and pupillary symptoms are absent. Sensory symptoms do not present themselves with any regularity. They consist of dull pains, slight anæsthesia, delay in sensory conduction, and hyperalgesia. Various deformities mark the later stages of Friedreich's ataxia. Lateral curvature of the spine is commonly present, and may or may not be associated with angular deformity. Talipes equinus or equino-varus frequently appears. A characteristic deformity of the feet is the dorsal flexion of the great toe on its phalanx.

FIG. 192.



The feet in Friedreich's disease.—After Bury.

(*c*) **Acute Myelitis.**—The etiology of acute transverse myelitis is found mainly in traumatic and infectious processes. In traumatic cases, the cause is self-evident. In the cases arising otherwise, it is not so clear. The toxæmia giving rise to it include the many acute infectious diseases, notably typhoid fever, scarlatina, measles, malaria, gonorrhœa, syphilis, erysipelas, and septic infection. Exposure, alcoholism, sexual excesses, inordinate exertion, must be relegated to the class of predisposing factors. The premonitory symptoms of acute myelitis are those of acute inflammatory affections generally, and include high temperature, rapid pulse, and the usual constitutional phenomena of fever. The initial spinal symptoms are those indicative of irritation, soon followed by others due to impaired function. It should be understood at the outset that the symptomatology of myelitis must vary with the level of the spinal-cord lesion, and the extent to which the same has involved the different sets of conducting tracts.

In the beginning the patient complains of pains and paræsthesiæ, the latter being unquestionably the more prominent. These paræsthesiæ include numbness, formication, and tingling, noticeable especially in the extremities. Pain, when present, is confined to limited areas; sometimes it is in the back, and sometimes it assumes the well-known girdle character. Severity and persistence of pains suggest that the inflammation has involved the meninges as well as the spinal cord itself. The motor symptoms of myelitis first appear as a sense of dragging or heaviness in the limbs, gradually increasing in intensity until it amounts to absolute paralysis. With this loss of power comes a loss of sensation, which likewise increases to complete anæsthesia. The time occupied by these symptoms in reaching their maximum intensity varies in different cases; it may be but a few hours in some, while in others it may not be for several days. In traumatic cases, when the cord has been greatly compressed, motor and sensory paralyses are present from the start. The inflammation and its special symptoms are secondary. Trophic phenomena are commonly present, and are very distressing. The skin usually suffers, and becomes dry and harsh. The temperature of the paralyzed limbs is nearly always higher than that obtained under the tongue. The most important lesion is the bedsores, which may occur on any part exposed to pressure, but especially are they likely to appear over the buttocks and sacrum. Even the light pressure of the heels on the bedding frequently produces them in that locality. The bladder and rectum rarely escape in acute myelitis. Usually there is incontinence of both urine and fæces. When an entire transverse segment of the cord is involved, the reflexes dependent upon the integrity of all segments below the one affected are destroyed. If, however, some fibres are still intact, these reflexes are preserved, or even exaggerated. The muscles receiving their nerve-supply from the anterior cornua in the affected segment undergo atrophy, and present the reaction of degeneration. When the cervical portion of the cord is the seat of the pathological process, all four extremities are paralyzed. In addition, there will be observed irregularity of respiration. The prognosis of acute myelitis is highly unfavorable. Death usually results from exhaustion within a short time. Those cases which recover become hopeless cripples.

(*f*) **Thrombotic Softening of the Spinal Cord.**—The possibility of the existence of thrombotic softening of the spinal cord has been seriously called in question by eminent authority. Cases have been observed, however, which place the disease beyond dispute. The symptoms, resulting, as they do, from lesions of the cord, must present features which are almost indistinguishable from those of acute myelitis. Etiological factors must constitute an important feature in establishing the diagnosis, *i.e.*, the patient must give evidence of vascular changes capable of giving rise to thrombosis, these including atheroma and syphilis in particular. In the only case in which the diagnosis of spinal thrombosis was made by me, the patient, an elderly man, had repeated attacks of paraplegia, each one worse than the preceding, and finally presenting the full clinical picture of complete transverse myelitis.

(*g*) **Hereditary Spastic Paraplegia.**—Cases belonging to this category are rare. My experience is limited to the observation of a series of cases studied and reported by Bayley. In them the line of hereditary tendency to this disease was remarkable, having been traced through no less than five generations. There were no special features attached to the spastic paraplegia in these cases other than those which one would expect to see in paralyses of like character first manifesting themselves early in life.

(*h*) **Reflex Paralyses.**—I have introduced the subject of reflex paralyses simply to indicate my lack of faith in the possibility of any such occurrence. Careful study will relegate all the alleged examples to either hysteria, myelitis, or the various simple and combined system diseases of the spinal cord. A belief in the existence of these palsies is a barrier to scientific clinical investigation. The doctrine of the origin of paralysis in reflex irritation was promulgated at a time when the pathology of nervous diseases was in a chaotic state. To believe in it to-day is a relic of barbarism.

(*i*) **Periodic Paralyses.**—Cases of this character are very rare indeed; but examples have been reported by competent neurologists. Their pathology is not at all understood.

Peripheral Paralyses.

Peripheral paralyses are recognized by their distribution, being confined to areas supplied by one or more nerve-trunks. The pathological lesions upon which they depend include neuritis and traumatism. The latter term is here used in its broadest sense, including external injury and compression within by reason of tumors, abscesses, etc.

Neuritis may be either simple or multiple, *i.e.*, limited to one nerve or nerve-trunk, or involving numerous nerves. In accordance with the general law that " Symmetrical multiplicity means a blood state acting alone, irregular multiplicity means a blood state combined with local determining causes, and purely local manifestation means a local cause," we expect simple neuritis to originate in local causes; hence we find it to be due to injuries, local exposure, pressure of tumors, and faulty postures. But its onset may be favored by constitutional predisposition, as it may result from syphilis, lithæmia, leucocythæmia, and rheumatism. In multiple neuritis, the constitutional origin of the cause is more apparent. We find this disease especially liable to occur after toxæmiæ, notably those of typhoid fever, diphtheria, diabetes, syphilis, tuberculosis, rheumatism, gout, variola, septicæmia, alcohol, lead, arsenic, and bisulphide of carbon.

The symptoms of simple neuritis must necessarily vary with the acuteness of the lesion and the functions of the nerve involved. Thus its onset may be rapid or slow. Pain is generally the first symptom to attract notice.

At first it is localized over the course of the affected nerve, and is greatly aggravated by pressure, and by influences which increase the tension on the affected nerve. Pressure upon the nerve sometimes excites pain, which extends to its utmost points of distribution. Sensory disturbances are likewise prominent. These consist of numbness, tingling, and anæsthesia in the regions supplied by the affected nerve. They may constitute the first symptoms, and in mild cases the only ones. They are generally experienced in the feet and hands. When the patient is thin, or the course of the nerve superficial, the inflamed and swollen structure may be felt. Constitutional symptoms are rarely prominent. Motor symptoms in the shape of paralyses are not likely to be obtrusive unless the traumatism giving rise to the inflammation has been a severe one. In most cases, however, careful examination reveals the presence of some loss of power. The paralysis partakes of the characteristics of that due to lesions of the lower motor neuron, *i.e.*, there is muscular atrophy, with the reaction of degeneration. Fibrillary contractions are associated with the advancing muscular atrophy. In the chronic forms of neuritis pain is the most prominent feature; indeed, these cases may be diagnosed as neuralgia.

In multiple neuritis the symptoms will vary greatly according to the nature of the case. Taking those originating in excessive alcoholic addiction as a type, we find a history of numbness, tingling, various paræsthesia, gastric symptoms, insomnia, tremor, and peripheral pains extending over a period of months. Finally the paralysis sets in with more or less rapidity. It usually starts in the lower extremities, affecting the anterior tibial group of muscles, thus producing the symptom known as "foot-drop." Sometimes it involves hands and feet simultaneously, producing wrist-drop in the former. The paralysis may extend in severe cases until the muscles of all four extremities are involved. It may, but rarely does, involve the trunk. Tactile and muscular sensibility are seriously interfered with. Severe pains are often experienced. The affected parts are decidedly hyperæsthetic. Both superficial and deep reflexes are destroyed in the paralyzed parts. Mental symptoms are nearly always associated with alcoholic neuritis, and include delirium, illusions, hallucinations, insomnia, loss of memory, and defective power of concentration. In women suffering from alcoholic neuritis there is nearly always an associated amenorrhœa. Urticaria and purpuric blotches on the lower extremities have been observed by Buzzard.

In multiple neuritis succeeding diphtheria, paralysis with anæsthesia is the prominent feature. There may be, as associated symptoms, paralysis of deglutition, double vision, or loss of the power of accommodation. Pains are not as prominent as they are in some other varieties of neuritis.

Post-typhoid neuritis exhibits both pain and paralysis. It is to be diagnosed by the history of the case.

Arsenical neuritis is characterized mainly by the prominence of the symptoms.

Plumbic neuritis involves mainly the musculo-spiral nerves.

Paralyses of the Upper Extremities.

These may result from injury of the brachial plexus as a whole or in part (plexor or combined paralyses), or they may be due to lesions of individual nerve-trunks.

Plexus Paralysis.—The patient cannot raise his arm at the shoulder, nor can he flex the forearm at the elbow; the movements of the wrist are unimpaired; and the arm when flexed at the elbow can be extended by the unaffected triceps. This indicates loss of power of the deltoid, biceps, infraspinatus, supraspinatus, brachialis anticus, and supinator longus, and is due to injury of the nerve-trunks from the fifth and sixth cervical roots. It is commonly known as Erb's paralysis, and is not infrequent as the result of injury during birth. It may also occur from neuritis, pressure of tumors, and constrained posture during ether narcosis.

Inability to extend the arm at the elbow, flex the wrist and fingers, and **pronate the forearm,** is due to lesions of the seventh and eighth cervical and first dorsal nerve-roots. It is known as lower plexus paralysis. Such cases have been produced by pressure of tumors, osseous thickening of the first rib, syphilitic meningitis, and neuritis.

Complete paralysis of the arm is known as total plexor paralysis, and is always traumatic in origin, resulting from injuries during parturition, dislocation of the humerus, or fractures of the clavicle or humerus.

The inferior angle of the scapula assumes a position nearer to the vertebral column than normal, and at the same time the posterior border of the bone projects, giving it an appearance that has been described as "winged." Such a condition is due to paralysis of the serratus magnus muscle, which is supplied by the long thoracic nerve. It is generally produced by compression, as in carrying heavy weights upon the shoulder, or by muscular action with the arm elevated, as in whitewashing a ceiling. It may also result from "cold." Paralysis of the serratus is also observed as part of general palsies, as in progressive muscular atrophy and acute poliomyelitis anterior.

Inability to raise the arm at the shoulder. This is due to paralysis of the deltoid and teres-minor muscles, which are supplied by the circumflex nerve. The functionless muscles undergo atrophy and there ensues flattening of the shoulder. The ligaments relax, and a concavity forms beneath the acromion process. Circumflex nerve paralysis is generally the result of blows on the shoulder, or crutch-pressure.

Inability to extend the hand and fingers, with paralysis of the supinator longus. In such a case there is disease or injury of the musculo-spiral nerve. This is probably the most frequently observed of the peripheral palsies. In a majority of cases it is the result of pressure of the head upon the arm while asleep,—generally, too, when the patient is in a state of alcoholic intoxication. The accident under such circumstances is made all the more serious by reason of the fact that the subject thereof already has a predisposition to neuritis because of his alcoholic saturation. Very exceptionally it may result from toxic causes, including typhoid fever, the puerperal state, and acute articular rheumatism. A very common toxic cause is found in neuritis from lead-poisoning. If the nerve happens to be injured high up in the arm, then the triceps will also be paralyzed. While the actual power of the flexor muscles is unimpaired, there appears to be a weakness of grasp of the affected hand. This is due to the fact that the origin and insertions of the flexors are unduly approximated, and hence cannot act to the normal advantage. The associated disturbance of sensibility is, as a rule, but slight, consisting usually of some slight formication and numbness.

Bilateral wrist-drop or double musculo-spiral palsy may result from simultaneous injuries of both nerves, as from pressure of rope when the subject has been lashed to a mast, or has fallen into a drunken sleep with both arms hanging over a sharp edge. The majority of cases of double wrist-drop are, however, the result of lead-poisoning. In such cases, there will be a history of other evidences of the untoward action of lead, as colic, blue line along the gums, etc.

Pronation beyond the mid-position is impossible; flexion at the wrist is still possible, but only with strong deflection of the hand towards the ulnar border of the forearm; the thumb rests extended and adducted; the second phalanges cannot be flexed on the first, and the distal phalanges of the first and second fingers on the middle phalanges. This state of affairs is due to paralysis of the pronators, the flexor carpi radialis, the flexors of the fingers, with the exception of the ulnar half of the deep flexor, the flexors and abductors of the thumb, and the first and second lumbricales. These muscles are supplied by the median nerve. Paralysis of this nerve almost always occurs in conjunction with other brachial palsies, as after luxations and the pressure of the Esmarch bandage. It may be injured singly by penetrating wounds of the arm. Cases have been reported in which it has been injured by forcible contraction of the triceps. Median nerve palsy is generally associated with pains, hyperæsthesia, and impaired sense of touch.

The **hand deviates to the radial side; flexion of the first phalanges is impossible, as is also adduction of the thumb.** The muscles paralyzed are the flexor carpi ulnaris, the inner part of the flexor profundus digitorum, the muscles of the little finger, the interossei, the third and fourth lumbricales, and the adductor and inner head of the short flexor of the thumb, and the nerve diseased or injured is the ulnar. Sensation on the ulnar side of the hand, more especially on the dorsum, is also impaired. This condition occurs in connection with other brachial palsies, as a result of luxation pressure. It may occur singly, as the result of fractures of the internal condyle of the humerus and supra-condyloid fractures. Sometimes it is produced by the pressure of the resulting callus. Cases have been observed in which it has followed slight pressure, as from resting the inner surface or the elbow or the internal condyle upon a hard support. It has occasionally resulted from prolonged flexion of the elbow.

In old cases the phalanges become hyperextended, while the others assume a position of strong flexion. The hand becomes clawed, producing the so-called "main en griffe."

Dana describes what he calls a "*symmetrical spontaneous ulnar neuritis*," a condition which develops slowly in neurotic subjects without known cause. It is characterized by pain and paræsthesia in the parts supplied by one ulnar nerve, with subsequent deformity and extension to the opposite side. He assigns degenerative neuritis as the pathological cause. Mild cases of this disorder are probably very common.

Paralyses of the Lower Extremities.

Loss of power of flexion of thigh on the abdomen and of extension at the knee. The ability to flex the thigh upon the abdomen depends upon the functional activity of the psoas, iliacus, and quadriceps extensor muscles. All of these, together with the sartorius and pectineus, receive their nerve supply from the anterior crural nerve. In order that all of these muscles shall be paralyzed, it is essential that the lesion of the nerve be situated at or near its origin in the lumbar plexus. The more common conditions existing in this location and capable of compressing the nerve include enlargement of the retroperitoneal lymph-glands, tumors of the vertebral column, and psoas abscess. An idiopathic neuritis involving the anterior crural is a rare condition, but cases have occurred as the result of gout and exposure to wet. Bilateral paralysis has resulted from alcoholic excesses. Traumatism limited to this nerve is exceedingly rare, owing to the well-protected situation of the nerve ; but cases have occurred as the result of direct wounds, and fractures of the thigh and pelvic bones. When the nerve is injured or diseased after emerging from beneath Poupart's ligament, loss of extension at the knee alone results. This is due to paralysis of the quadriceps extensor. While the sartorius and pectineus are also involved, the symptomatology of the case is not complicated thereby, because paralysis of these muscles produces no symptoms.

Loss of power of adduction of the thigh and inability to throw one leg over the other; impaired outward rotation of the thigh. Impaired function, as indicated above, results from injury or disease of the obturator nerve. It is a rare condition, but may result from difficult parturition, pelvic tumors, or obturator hernia.

Inability to rotate the thigh outward, with difficulty in gaining an erect posture after stooping, and in rising from a sitting position. This depends upon disease of the superior gluteal nerve, which, however, is seldom paralyzed alone. The muscles to which it is distributed are the gluteus medius and minimus and the tensor vaginæ femoris. The superior gluteal nerve is usually injured in conjunction with the lumbo-sacral cord, in parturient accidents.

Impairment of the power to rise from a sitting position, with normal ability to stand upright. This motor defect arises from paralysis of the gluteus maximus, which is supplied by the small sciatic nerve. It is rarely involved, apart from other nerves.

Loss of power of flexion of the leg on the thigh and complete paralysis of all the muscles below the knee. This is, in short, the condition observed in cases of a complete transverse lesion of the great sciatic nerve above the middle third of the thigh. When the lesion is below that point, the leg-muscles only are paralyzed. Walking is not as much interfered with in this form of paralysis as one might imagine. The limb is held rigid at the knee by the quadriceps extensor. In walking, the limb is lifted by what might be termed hyper-flexion at the hip. The nerve-lesions capable of producing sciatic nerve palsy include, for the most part, tumors and injuries.

Neuritis always impairs the muscular power in the area of distribution; but the pain in such cases is so prominent that other symptoms escape observation.

Loss of power of extension of the foot upon the leg and of extension of the first phalanges of the toes. Such depreciation of function as this depends upon the paralysis of the tibialis anticus, the long extensor of the toes, the peronei, and the extensor of the little toe, all of which muscles are supplied by the external popliteal or peroneal nerve. This is one of the types of palsy characterized by what has been so graphically described as "dropped-foot." As the ankle cannot be flexed, the whole leg is lifted higher than normal in walking, the toes at the same time drooping. This has been called "the steppage gait." The peroneal nerve is rarely paralyzed alone. Owing to its exposed position, it may be injured. When paralysis occurs in its distribution it is nearly always as a part of a general neuritis, as that resulting from alcohol or the infectious diseases.

Loss of power to flex the foot upon the leg, and of flexion of the toes; inability to rotate the foot inward. Paralysis of this character is dependent upon loss of function of the popliteus, the calf-muscles, the tibialis posticus, the long flexors of the toes, and all the muscles of the sole of the foot; and these are supplied by the internal popliteal or posterior tibial nerve. The position of the foot is that of talipes calcaneus.

The toes become flexed at the last two joints and extended at the others. This position is due to disease of the external plantar nerve. It interferes very greatly with walking.

While on the subject of paralysis, reference may be made to two conditions which exhibit motor insufficiency as a prominent feature, but which are not paralytic in character, namely, "astasia abasia," and "intermittent claudication" or "intermittent limping." The latter has, associated with the motor insufficiency, severe paroxysmal pain, which is to be regarded as the important symptom. An extended description of the complaint will therefore be presented under the painful affections of the lower extremities.

Astasia abasia is a term used rather indiscriminately to indicate two separate and distinct conditions. One of these is certainly hysterical. The character of the other class of cases remains thus far undetermined.

In the hysterical variety the patient is unable to walk or stand, and yet she is able to move her limbs perfectly when recumbent. Sensation is likewise unimpaired. The hysterical origin of such a combination of symptoms is made all the more evident by their association with the stigmata of that disease.

The astasia abasia described by Gowers seems to be an entirely different affection. He allies it with vertigo, describing it in the following words: "The sufferer from it, when walking, will suddenly fall to the ground (astasia), often forward; it seems to him that his legs give way. When sitting, he will suddenly bend forward, his head dropping on the chest, and his body seems to lose its power of support (abasia). In either case, in a minute or two, normal strength returns. There is never loss of consciousness." "Occasionally the fall has been preceded by a sensation as if struck by a hammer on the back of the neck. It occurs chiefly in persons past middle life, and in those who have degenerated blood-vessels." The disease seems to be akin to, if not identical with, an affection described by Searle, of Brooklyn, many years ago, and known for some time afterwards as "Searle's disease."

Motor Excitements (Hyperkineses).

These include a great variety of symptoms, the main characteristics of which are increased or abnormal muscular contractions. They are convulsions, spasms, rigidities, contractures, choreic and automatic movements, and tremors. It is now in order to study these symptoms *seriatim*.

Convulsions.—A convulsion may be defined as a condition characterized by more or less widespread muscular contractions and relaxations in rapid alternation, or by prolonged muscular contractions. The term "spasm" is used to designate convulsive movements which involve but a limited portion of the body.

A **tonic convulsion** or spasm is one characterized by prolonged or sustained muscular contraction; in other words, it is a convulsion characterized by rigidity.

A **clonic convulsion** is one in which muscular contractions and relaxations succeed each other in rapid succession.

Classification of Convulsions.—Clinically, we may divide convulsions into three varieties, as follows:

A. Epileptiform.
B. Hysteroid.
C. Tetanic.

(*a*) **Epileptiform convulsions** are so-called because they constitute the typical variety of convulsive movements observed in epilepsy. Still, it must not be forgotten that they may be observed in a variety of other conditions. The essential features of an epileptiform convulsion are the tonic, followed by clonic, spasms. At the same time, the paroxysm may be preceded by certain symptoms known as the aura.

Epileptiform convulsions may occur in

a. Epilepsy.
b. Hysteria.
c. Organic disease of the brain.
d. From reflex irritation.
è. From certain blood states, as uræmia, lithæmia, etc.

(*a*) **Epilepsy.**—Typical epilepsy is a chronic disorder of the nervous system characterized by paroxysms of convulsions, consisting of tonic and clonic spasm, and associated with unconsciousness. The interparoxysmal period presents no evidence of ill-health unless it be some degree of mental impairment. The typically complete case of epilepsy, consisting of several stages, presents attacks as follows: The aura, the cry, the stage of tonic spasm, the stage of clonic spasm, and the stage of stupor. During all these stages, with the exception of the first, the patient is unconscious. One or more of these may be absent, and yet the seizures be none the less epileptic. The aura is a subjective sensation, motor phenomenon, or psychical manifestation immediately preceding, so as to almost form part of the epileptic attack. The convulsive seizure is ushered in by a cry, which is due to the sudden tonic spasm of the muscles of expiration forcing air through the larynx, which is also contracted in tonic spasm. The tonic spasm comes on so suddenly that the patient has no opportunity to reach a place of safety before he falls unconscious. The entire body is rigid, sometimes in one position, sometimes in another. The rigidity of the muscles of respiration usually brings on a cyanotic condition. The pupils are insensitive and widely dilated. In the course of thirty seconds to a minute and a half the tonic spasm gradually disappears, and the clonic spasm begins. The movements now assume a decidedly shock-like or jerking character. Involving the muscles of the thorax, saliva is forcibly and intermittently ejected from the mouth by the air, and so become frothy. After a period ranging from one to five minutes, this stage disappears,—not by the individual movements of which it is composed becoming less strong, but by their frequency diminishing. Then ensues the stage of stupor from which the patient may sometimes be aroused, though with difficulty. This stage may continue for a number of hours. It is not uncommon for urine and fæces to be discharged involuntarily during the paroxysm; neither is it uncommon for the tongue to be caught between the teeth during the spasm, and thus be badly bitten. The patient, being fully over his convulsion, is in a condition of apparently good health; *i.e.*, he is free of all objective and subjective evidences of disease. The important data upon which a diagnosis of epilepsy is based include the suddenness of onset of the paroxysms without assignable cause, the absolute loss of consciousness, the short duration of the convulsive period, and the good condition of health between the attacks.

At present but little can be said concerning the etiology of epilepsy. There can be no doubt that the many cases which we now diagnose as belonging to the class under study are in reality due to a variety of conditions. The term "epilepsy," therefore, is a generic one. In many instances neurotic inheritance and developmental errors are important etiological factors. Traumatism, hereditary syphilis and the convulsive habit are also important though not well understood causes.

(*b*) **Hysteria.**—Although convulsions of epileptiform type are by no means the characteristic form observed in hysteria, nevertheless they may occur in that disease and make the diagnosis a very difficult problem. So far as the disorderly movements are concerned, they differ in no particular from those of true epilepsy. But if the entire attack be observed carefully, important distinctions will arise. The patient, practically always a woman, falls unconscious and goes at once into tonic, rapidly succeeded by clonic, spasms; and the latter, in turn, is succeeded by phenomena well-known to be hysteroid. The only possibility of diagnostic error lies in the physician's knowledge of the patient being limited to what he observes during the seizure.

In hysteria, the symptomatology of the interparoxysmal period is of the highest diagnostic importance.

(*c*) **Organic Disease of the Brain.**—The typical convulsions of organic brain disease are of the so-called Jacksonian type ; *i.e.*, the convulsions are limited in their distribution, are not attended by loss of consciousness, and are succeeded by paralysis of the convulsed parts. In some cases, consciousness is lost. Convulsions of this type suggest the presence of a lesion in the cortical motor areas. Then we find another class of cases in which the convulsions are general, and, so far as the movements themselves are concerned, are exact counterparts of those occurring in epilepsy. The attacks in these cases are much longer in duration than are those of epilepsy. The patient is usually advanced in years, and between the seizures there occur headaches, vomiting, vertigo, optic neuritis, paralysis, and other symptoms of cerebral disease. The interparoxysmal symptoms will vary, of course, with the nature of the pathological changes, *i.e.*, whether the lesion be tumor, abscess, hæmorrhage, degeneration, embolism, thrombosis, or acute or chronic inflammation.

(*d*) **Reflex Convulsions.**—Herein we have a most interesting and important clinical subject. To judge from carelessly-made remarks, one would incline to the belief that reflex convulsions are of common occurrence. Analytical study of cases demonstrates that, if we except the cases occurring in infancy and early childhood, reflex convulsions are to be included among the rare clinical conditions. The unfortunate part of the whole matter lies in the fact that, as a result of having convulsions diagnosed as reflex, patients are subjected to a great variety of operative treatment, said treatment varying according to the prejudices of the operator, including ocular tenotomies, circumcisions, rectal and gynecic procedures in great varieties, to say nothing of the fads of dietetics. It is a good rule to follow, in the case of a convulsion occurring for the first time after the third year of life, to consider the probability of its being epileptic as very strong, indeed. Convulsions prior to that age are, in by far the majority of cases, either reflex in origin or symptomatic of organic disease. Reflex convulsions nearly always give a history which points out the seat of the causative irritation. In the case of a cicatrix, we should expect it to be sensitive to pressure, etc. When the attacks are dependent upon uterine or ovarian irritation, symptoms referred to those organs establish the relation of cause and effect indubitably. So far as the character of the attacks is concerned, reflex convulsions, as a rule, simulate those of epilepsy so closely as to generally make a differentiation impossible. The reflex seizures are not, perhaps, as sudden in their onset, and in them the disturbance of consciousness is not as profound as in epilepsy. Their duration is generally longer. Epileptic seizures usually subside in five, or, at most, in ten minutes ; reflex convulsions are of indefinite duration. When one reflex spasm has occurred, it is very apt to be followed shortly by others in rapid succession. In the case of epilepsy, the patient is apparently well after the attack, and the second paroxysm does not follow for a long period. Having diagnosed reflex convulsions, it is always a wise plan to doubt one's diagnosis, and care for the patient as if he might possibly become epileptic. Another important point, and one adding to the difficulties of the situation, is that the repeated occurrence of convulsions from reflex irritation may lead to the formation of the convulsive habit, and thus the disease may finally degenerate into a typical epilepsy.

(*c*) **The convulsions produced by various toxæmia** are distinctively of the epileptiform type. They differ from those of epilepsy, however, in their prolonged duration only. The class of cases most frequently observed in practice is that occurring in children as part of the initial phenomena of the acute infectious diseases, notably in scarlatina, diphtheria, intermittent fever, etc.

Next in frequency come the **uræmic** convulsions. For the most part they are general in their distribution. They are to be diagnosed by the history of the case and the associated phenomena. The latter include scanty urination, albuminuria, the presence of tube-casts, hypertrophy of the heart, increased vascular tension, and thickened arteries. An uncommon type of convulsions from renal disease, notably from interstitial nephritis, is found in Jacksonian convulsions, which may simulate those arising from brain disease so closely as to make the diagnosis a mere matter of conjecture.

The possibility of **lithæmia** producing epileptiform convulsions I very much doubt. Careful study of the alleged cases nearly always demonstrates that the paroxysms have been produced by other conditions than uric acid poisoning.

(B) **Hysteroid Convulsions.**—The view that hysteroid convulsions occur in hysteria only, as so long taught by eminent authority, does not seem tenable, if I am to judge by personal experience. In a number of instances of organic diseases of different kinds have paroxysms of this nature been present, the diagnosis being proven subsequently on post-mortem examination. A satisfactory explanation of the phenomena has not been offered. The only suggestion that I can make is that the organic lesions may have awakened a latent hysterical temperament into activity.

The characteristic hysteroid paroxysms consist of co-ordinated movements; to the laity they are best described as "struggling," as distinguished from epileptic spasms, which are "jerking." In their mildest form they may appear only as slight rigidity, following possibly some emotion, unpleasant or otherwise. In other cases they are manifested only by "globus hystericus," or a few purposive movements. While ushered in suddenly, the patient never falls so as to hurt herself. She manages to reach a place of safety to let herself down easily. Sometimes, however, her calculations go wrong, and she hurts herself accidentally. Some degree of apparent unconsciousness is nearly always present, but it is readily seen that this is not profound. Possibly some few clonic movements may appear. In still other cases the patient simply clutches at her clothing and surrounding objects.

The severe paroxysms of hysteria are usually referred to as hystero-epilepsy. They are seen in their best developed form in France, rarely in England and this country. The positions assumed by the patient are both grotesque and theatrical. At one moment bent in a position of extreme opisthotonos, the body resting on the forehead and toes, and at another making the wildest bounding movements; now assuming expressions of pleasure, and the next of extreme depression or terror; now exhibiting religious fervor, and then becoming erotic or obscene; now rolling about the bed, and then lying in perfect quiet; a picture certainly alarming to the uninitiated.

The severe paroxysms, as seen in America, are often ushered in by an aura, consisting usually of the globus hystericus, palpitation of the heart, or the bilateral foot aura. They may appear spontaneously, or as the result of some violent emotion. They are often ushered in by a loud cry, which very often continues throughout the paroxysm. This cry may consist of a mere shriek, or of words appropriate to some emotional state. Sometimes the patient talks considerably during the paroxysm, the words generally being mumbled, and without any connected meaning. Opisthotonos is very often present. It not infrequently appears with the patient lying on her side.

The differentiation of hysterical from epileptic seizures has well been tabulated by Gowers, as follows:

	Epileptic.	Hysteroid.
Apparent cause.	Absent.	Emotional disturbance.
Warning.	Any, but especially unilateral, or epigastric aura.	Palpitation, malaise, choking, bilateral foot aura.
Onset.	Commonly sudden.	Often gradual.
Scream.	At onset.	During course.
Convulsion.	Rigidity, followed by "jerking," rarely rigidity alone.	Rigidity, or "struggling," throwing limbs and head about.
Biting.	Tongue.	Lips, hands, and more often other people and things.
Micturition.	Frequent.	Never.
Defecation.	Occasional.	Never.
Duration.	A few minutes.	Often half an hour, or several hours.
Restraint.	To prevent accident.	To control violence.
Termination.	Spontaneous.	Spontaneous, or artificial (water etc.).

The distinctive movements in hysterical convulsions are the coördinated. These simulate voluntary movements. In a general way, they may be described as "struggling." The patient squirms, stretches, strikes, clutches at her hair, tears at her clothing, bites herself and others, kicks, strikes her head against the floor, etc. These movements are repeated with the utmost irregularity. Exceptionally, one or two movements are selected and repeated rhythmically. It is not unnatural that these attacks should terrify the bystander and lead to strong efforts to restrain the patient —efforts which almost invariably only intensify the convulsions. Almost certainly can we rely upon it, when we are told that it required several persons to control the patient because of the violence of the attacks, that the convulsion was hysterical in character.

(C) **Tetanic Convulsions.**—Tetanic convulsions are characterized by prolonged rigidity. In other words, they consist of tonic spasm only. Characteristic features of the type are extreme rigidity of the body. When with this rigidity there is arching backward of the head and neck, with bowing of the body, we have what is known as opisthotonos; when the arching is in the direction the reverse of opisthotonos, we call the condition emprosthotonos; arching to one side is known as pleurosthotonos.

Tetanic convulsions are observed in the following diseases:

a. Tetanus.
b. Hysteria.
c. Strychnia poisoning.
d. Tetany.

(*a*) **Tetanus.**—Prolonged tonic spasms are characteristic of tetanus; hence the use of the word "tetanic" as describing the type. It is now universally acknowledged that tetanus is the result of a specific infection, although cases may be observed in which it is impossible to trace such an origin. The first symptom noticed is usually a tonic contraction of the muscles of mastication, making it difficult or impossible to open the mouth (lock-jaw; trismus). Almost at the same time there appears a rigidity of the muscles of the back of the neck. These symptoms increase in intensity, the more rapidly, the more severe the case. It may happen that the rigidity of the back and neck and the trismus interfere so little with the patient's duties that he may go about his affairs without suspecting the serious nature of his illness. Ordinarily, however, the sufferings are too intense to permit of this. In severe cases, the opisthotonos is so extensive that the rigid body forms an arch, and it is said that it may be so extreme that the head and heels touch, the body thus being made to form a complete circle. As the disease progresses the spasms extend to the limbs. The convulsions in tetanus, with the exception of the trismus, are not constant; they come in paroxysms, which are renewed from the stimulus of slight peripheral irritation, as light, sound, or touch. The duration of the attacks will vary from a mere momentary jerking to a prolonged spasm, which may continue for several hours. The muscles of respiration usually participate, as exhibited by dyspnœa, short, quick breathing, and sense of suffocation. The face presents the characteristics known as the *risus sardonicus*, owing to tonic contraction of all the facial muscles. The eyebrows are elevated by the occipito-frontalis, the palpebral fissure is lessened by the contraction of the orbicularis palpebrarum, and the angles of the mouth are distorted by the muscles attached to the orbicularis oris. In a variety known as *facial tetanus*, the muscles supplied by the facial nerve on one or both sides are paralyzed, giving rise to a peculiar state of affairs, for which no satisfactory explanation has been offered. There is no definite rule respecting fever in tetanus. Many cases run an entirely afebrile course, while in others the temperature may rise to 100° F. or more. Chronic tetanus has been described; but it is not a separate clinical entity; it is simply a tetanus running a prolonged course.

Tetanus is always a serious disease, the vast majority of cases ending fatally. Death may occur in the course of the spasms at a time when the patient is believed to be progressing favorably.

(*b*) **Hysteria.**—The tetanic spasms in hysteria in the beginning cannot, excepting from the history of the case, be differentiated from those of tetanus. Soon, however, cerebral symptoms and the hysteroid movements appear, and the nature of the case is plain.

(*c*) **Strychnia Poisoning.**—The history of the case is invaluable in differentiating strychnia poisoning from tetanus. But this is not always available. The initial symptoms in the former include a general restlessness with hyper-acuteness of the special senses. Then the tetanic spasms appear and develop very rapidly, involving the entire body simultaneously. They differ from those of tetanus, however, in that trismus is the last symptom to appear, and the first one to disappear should the case recover. As in tetanus, consciousness is unimpaired almost to the end. The slightest external impression is sufficient to cause a renewal of the spasms.

(*d*) **Tetany.**—The spasms in tetany are always symmetrical, and involve the hands and feet first. In mild cases, and in those occurring in children with rachitis, they do not extend any further. The fingers are flexed at the metacarpo-phalangeal joints, and extended at the phalangeal, while the thumbs are drawn into the palms. The wrists are slightly flexed. If the shoulder-joints are involved, the arms are held in rigid adduction. The feet are extended and inverted, and the toes strongly flexed. The knees are rigidly extended. In severe cases, the rigidity extends to the muscles of the trunk and face. Very exceptionally, the jaws are rigid. The angles of the mouth may be drawn, exposing the teeth. It is even possible for the muscles of the eyeballs to become affected, in which case we have strabismus. The muscles of the tongue, pharynx and larynx are seldom affected; yet their implication is not impossible. The duration of these spasms presents the greatest variations, ranging as it does all the way from fifteen minutes to several weeks. They may be continuous or intermittent. They may continue during sleep, though generally mitigated somewhat during that period. They may even begin during sleep. When the contractions are severe they excite pain. The temperature is usually normal, though it may be elevated,—rarely, however, to more than 101° F. A very interesting phenomenon observed during the interparoxysmal period is that known as Trousseau's symptom. If pressure be made for a number of seconds on the artery or one of the main nerves of an affected limb, the spasm is re-excited. This is regarded by most authorities as a very important diagnostic symptom. By others it is not looked upon as constant. The electrical excitability of nerves and muscles is greatly increased to both faradic and galvanic currents. The mechanical irritability of the muscles is likewise increased; pinching or stroking them serves to bring on a contraction.

Local spasms occur in

a. Organic diseases of the brain.
b. From reflex irritation.
c. Hysteria.
d. From "habit" (so-called habit-spasm).
e. Cerebral and spinal meningitis.
f. The various occupation neuroses.

(*a*) **Organic Diseases of the Brain.**—As already stated in a previous paragraph, localized convulsions of tonic and clonic character occur as the result of irritative lesions of the cortical motor area, *i.e.*, in or about the convolutions bordering on the fissure of Rolando. The abnormal movements will involve the arm, or leg, or face, according to the particular portion of the motor cortex involved. Thus if the lesion be limited to the upper portion, the leg on the opposite side of the body is affected; if the middle portion, the arm; and if the lowest third, the face. Aside from these convulsions, we may also observe, in connection with organic brain diseases, such spasmodic phenomena as athetosis and jerkings or twitchings.

(*b*) **Reflex Irritation.**—In reflex irritation, I believe we find an important cause of some cases of local spasm; but the doctrine of reflex origin of such cases must not be carried too far. The special portions of the body demanding investigation as to the cause of the trouble include the eyes (for refractive and muscular anomalies), the nose (for spurs and other causes of obstructed nasal breathing), the teeth (especially in spasms of the tongue and facial muscles), the uterus and ovaries (for obscure spasmodic affections in women not clearly traceable to hysteria), and the urinary organs (in cases of spasmodic or involuntary escape of urine). However important the removal of sources of local irritation may be, it is important to remember that one must not search for them as a "fad." The imagination of the examiner must not be forced to make their discovery. It is unlikely in the highest degree that any lesion is capable of producing reflex symptoms unless it is of itself sufficiently obtrusive to produce symptoms directly referable to it, in which case its local treatment is necessary for its own sake. Much harm has been done by disregard of this teaching. Patients without limit have been subjected to ocular tenotomies, nasal cauterizations, tooth extractions, oöphorectomies, hysterectomies, and anal dilatations, to cure lesions existing only in the too active imagination of the "faddist."

(*c*) **Hysteria.**—The localized spasms symptomatic of hysteria present no typical clinical features. Many times have I seen them present characteristics which made their differentiation from organic cerebral disease most difficult. Even in such cases, careful investigation will disclose some one or more particulars in which they differ materially from the spasms of organic cerebral diseases, while the interparoxysmal period will disclose the presence of some well-known hysterical stigmata. Before a diagnosis of hysteria can be established, however, it is essential to eliminate the possible presence of symptoms indisputably the result of organic diseases, including in this category pupillary disturbances, inequality of the deep reflexes, optic neuritis, etc.

(*d*) **Habit-Spasm.**—The term "habit-spasm" is an unfortunate one, as the class of spasmodic affections given this name are not due to "habit," though they truly become such by reason of their long continuance. Each case of the disorder presents features peculiarly its own. Thus, in one patient there are spasmodic movements of the eyelids or winking movements; in another, jerking of the face; and so we might multiply the clinical types according to the particular region of the body involved. In some of the cases the abnormal movements continue for a month or two and then disappear, while in others they persist, despite treatment, throughout life.

(*e*) **Cerebral and Spinal Meningitis.**—Under this heading may also be included other than the purely inflammatory lesions of the meninges. The spasmodic movements are the result of the irritation of nerve-roots by the irritation of the tumor or the inflammatory changes, the abnormal movements occurring in the muscles to which the said nerve-roots are distributed. The diagnosis in these cases is to be made by associated symptoms, which point to meningeal involvement.

(*f*) **Occupation Neuroses.**—Under this name is included a large class of nervous affections characterized for the most part by spasm of incoördination of certain muscles used habitually in the pursuit of the patient's occupation; said spasm or incoördination occurring, as a rule, when the act which gave rise to the trouble is performed, and not during other movements of the muscles, no matter how complicated they may be. Of the occupation neuroses, the best known are writers', telegraphers', musicians' and sawyers' cramps. We sometimes designate these affections "palsy," using a qualifying adjective for each occupation. All cases of occupation neuroses do not exhibit spasm, for in some the prominent clinical features are tremor, paralysis, or neuralgia. The onset of the affection may be either sudden or gradual, usually, however, the latter. The patient notices that he cannot grasp his pen with his usual facility, and this leads him to hold it all the tighter. Gradually this difficulty increases, and he, to obviate it, adopts quite a variety of devices in the way of unusual positions when writing. Finally there occur spasms of the muscles concerned in pen-prehension as soon as that instrument is taken in hand; consequently it slips from between the fingers. The spasm is always of a tonic character, and may be associated with some tremor. During the act of writing the pen is jerked about, imparting to the chirography the most irregular characteristics. Sensory disturbances may be associated. These include pain, sense of tiredness, and even neuralgia. It is said that the occurrence of pain indicates a co-existent neuritis. Sometimes there are vaso-motor phenomena, as " dead fingers," passive congestion of the hand and arm, local sweatings, and dryness of the skin.

Frequent forms of local spasm, according to distribution, include

a. Œsophagismus.
b. Spasmodic vomiting.
c. Phantom tumor.
d. Nervous cough.
e. Vaginismus.
f. Spasm of the vesical and rectal sphincters.
g. Asthma.
h. Facial tic.
i. Trismus.
j. Spasmodic torticollis (wry-neck).
k. Rhythmic or functional spasm.

(*a*) **Œsophagismus.**—This affection, or, more properly speaking, symptom, may be either primary or secondary. In the former case it is a local expression of a neurotic constitution, especially when occurring in young women. In this connection it may result from purely psychical states,—as, long after a foreign body has been swallowed and removed. Cases have also risen apparently from reflex irritation starting in distant organs, as in the genitals and the gastro-intestinal tract. As a secondary condition, it is commonly associated with most organic affections of the œsophagus, as inflammation and ulceration. It also occurs as one of the symptoms of hydrophobia. The special symptom which suggests the presence of spasmodic stricture of the œsophagus is dysphagia, which is more or less paroxysmal, and efforts at swallowing are attended by gulping noises. The difficulty seems to be increased by attempts at swallowing cold food. The patient does not emaciate, as in the case of organic stricture. The true nature of the obstruction is recognized by the œsophageal bougie, which at first meets with an obstruction. The latter yields, however, to continued mild pressure or to anæsthesia.

(*b*) **Neurotic Vomiting.** — The clinical features of vomiting arising from neurotic causes vary quite considerably. Prominent among such cases stands the so-called hysterical vomiting. Of course it is due to spasm of the stomach, which, in turn, may succeed a morbid sensitiveness of the parts. It is especially liable to occur in girls just past the age of puberty, and in young women. It seems to be induced by the taking of food into the stomach, and is almost invariably painless. In the majority of cases a portion of the food only is ejected; hence emaciation is not as prominent as the obstinacy of the vomiting would lead one to expect. Exceptionally, however, all food is vomited, and emaciation becomes profound, and may even have a fatal termination. This type is sometimes referred to as idiopathic nervous vomiting.

Another type of nervous vomiting is known as *juvenile vomiting*. It occurs among overworked school-children, and is attended by cardialgia, severe headache, pallor, slow pulse, and dilated pupils. The paroxysms of vomiting recur at rather frequent intervals, *i.e.*, every day or twice daily.

Periodical vomiting is also undoubtedly of neurotic origin. It occurs in apparently healthy persons, and the attacks recur with remarkable periodicity. The paroxysms continue for a variable period, generally from one to ten days. During their continuance the gastric irritability is extreme, and when they subside the stomach becomes at once tolerant of all kinds of food.

(*c*) **Phantom Tumors.**—Phantom tumors occur rather frequently among hysterical women. They are probably more common than ordinarily believed; hence it is important that the possibility of their existence should be excluded before proceeding with an abdominal section for their removal. The commonly accepted belief respecting their nature is that they are dependent upon a localized pneumatosis due to spasmodic contraction of the intestinal muscles, but they more frequently result from muscular contractions. They may occur in any portion of the abdomen, but are especially common in the lower portion, when due to a localized pneumatosis; and then they may be differentiated from solid growths in that they are tympanitic on percussion, and disappear under general anæsthesia.

(*d*) **Nervous or Hysterical Cough.**—The most commonly observed type of nervous cough is one characterized by frequently repeated hacking sounds, as if an attempt was being made to rid one's self of an irritation in or about the suprasternal fossa. In some individuals such a cough seems to be an analogue of the well-known habit-spasms. "Habit-cough" is not a bad name for them.

The true hysterical cough is paroxysmal, and is attended by laryngeal spasms; ofttimes, also, by other hysterical phenomena. It is especially liable to succeed some emotional agency.

(*e*) **Vaginismus.**—Spasm of the vagina may be idiopathic, *i.e.*, dependent upon the hysterical temperament, or it may be secondary to local lesions producing irritability of the parts. In many instances, general and local causes coöperate. The hysterical cases are recognized by the absence of local changes to account for the morbid sensitiveness, and their association with the well-known stigmata of hysteria. Those of local origin are generally observed in recently-married women, and are dependent upon small ulcerations or fissures about the hymen or its attachments. Vaginismus is frequently attended by spasm of the sphincter ani.

(*f*) **Spasm of the Sphincters of the Rectum and Bladder.**—The remarks just made respecting vaginismus apply with equal force to spasm of the rectal and vesical sphincters. From a clinical standpoint, the importance of these conditions has not been recognized. Spasm of the sphincter ani is an important cause of constipation, and may, if long continued, give rise to important changes in the rectal structures. On the other hand, it must be remembered that the rectal lesions may be primary and the anal spasm secondary. The influence of this fact on therapeutics is obvious.

(*g*) **Asthma.**—Nervous asthma has already been studied in full under Disturbances of the Respiratory Apparatus.

(*h*) **Convulsive Tics.**—Under this title is included a large number of cases characterized by sudden arrhythmic and involuntary contractions of muscles,—chiefly, however, in the muscles of the head and face, though in many cases those of the neck, arms, trunk and legs also participate. It is possible for the patient to restrain the movements for a time by a strong effort of the will, but such effort is exhausting, and produces sensations of great distress. The movements are first noticed in the muscles of the face; later, speech is involved. The grimaces thus occasioned are remarkable, sometimes simulating laughter and pleasure, at other times pain and misery. The natural course of the disease is from bad to worse, interrupted, however, by periods during which the progress is at a standstill. Finally the mind becomes involved, and it is not unusual to see insanity as the termination.

Patients with tic exhibit a remarkable tendency to imitate. Thus they repeat sounds and words heard (*echolalia*), gestures and movements (*echokinesis*), or they may exhibit uncontrollable impulses to ejaculate obscene words (*coprolalia*). There is inability to fix the attention, hence prolonged or consecutive thought is impossible. Ofttimes they are possessed of the most strange ideas, leading to correspondingly strange conduct.

Remarkable types of tic are observed in certain geographical sections, as "latah" in Malay, myriachit in Kamtchatka, and the "jumping Frenchman" about Moosehead Lake, in Maine.

(*i*) **Trismus.**—The presence of trismus, or lock-jaw, in tetanus has already been described, and need not be further referred to in this place. But trismus may occur in other conditions, as follows: Cerebro-spinal fever; dentition, especially during the eruption of the wisdom-teeth; as part of the clonic spasms of epilepsy, hysteria, abscess beneath the masseter and temporal muscles, tetany, and in uræmic convulsions. In each case the associated symptoms point unerringly towards the source of the trouble. The knowledge that these several affections can produce lock-jaw saves the physician from making a diagnosis of tetanus.

(*j*) **Opisthotonos. Emprosthotonos. Pleurosthotonos.**—The relations of these symptoms to tetanus have also been described. But they may likewise occur in other diseases, notably in hysteria. In this affection they may be even more marked than in tetanus; but then they are practically always associated with the wild coördinated movements of hysteroid seizures. They may also occur in strychnia poisoning, spinal meningitis, and uræmia.

(*k*) **Spasmodic Wry-Neck.**—The term "torticollis" is used to designate a variety of conditions in which an abnormal position of the head in its relation to the trunk is produced by muscular contractions. There is also a form of malposition dependent upon disease in other than the nervous and muscular structures, generally upon disease of the cervical vertebræ. To this the name "*false torticollis*" has been applied. Its recognition is very important because of the therapeutic indications thereby furnished. We have also the so-called "*rheumatic torticollis*," caused by acute myositis, and this is characterized by pain and tenderness of the muscular substance. "*Congenital torticollis*" is a variety in which the deformity is dependent upon permanent shortening of a muscle, usually the sterno-mastoid. It is believed to be due to intra-uterine or obstetrical injury of the sterno-mastoid, as it occurs most frequently in breech presentations. A peculiarity of this variety is its remarkable tendency to association with facial asymmetry. The affected muscle is found to be hard and firm, and the essential muscular elements more or less atrophied. "Spasmodic torticollis" is the name given when the deformity depends upon instability of nerve-cells. It is of two varieties, tonic and clonic. In the former the spasm is persistent and prolonged, assuming the appearance of a contracture. It is limited to one side, and usually to one muscle. When the sterno-mastoid is involved, the face is rotated to the opposite side, the chin somewhat elevated, and the head inclined to the affected side. When the trapezius is affected, the inclination of the head is more marked, the head is drawn backwards, and the shoulder is raised. Permanent contracture ensues in cases of long standing.

The clonic variety is of varied distribution. It involves one muscle or many; those of one side or both. Beginning in one of the muscles supplied by the spinal accessory nerves, it may extend and be widespread. The spasms may be few in number, occurring at long intervals; or, again, they recur in such rapid succession as to be disabling.

Prominent among the causes of spasmodic torticollis is the neurotic constitution. This is exhibited by the presence of cases of other nervous diseases, as epilepsy, migraine, etc., in different members of the family, and by a personal history of other neuroses in former years. Women seem to be affected with greater frequency than men. The disease is rarely observed prior to middle life. Cases occurring in women of less than thirty years of age are, in all probability, of hysterical origin. Injury of the parts, depressing emotions, otitis media, excessive use of the muscles, and malaria, have been assigned as causes in individual cases. It is very rarely, indeed, that it occurs from peripheral irritation.

The pathology of the disease is not at all understood. In no case has an autopsy revealed a lesion explaining the symptoms. It is believed that the morbid movements depend upon the overaction of nerve-cells. As to the situation of the nerve-cells, we must rely wholly upon conjecture. It has been observed that in some cases the muscles affected are those physiologically associated in their functions. In such it is fair to presume that the affected cells are of the higher centres, *i.e.*, the cortical. When no such association is found, the lower centres are the seat of disease. As to the nature of the changes which produce the overaction of the nerve-cells we must express complete ignorance.

SYMPTOMS.—Clonic spasms of definite distribution, but varying according to individual cases, comprise nearly the entire symptomatology of spasmodic torticollis. Usually spasm is the first symptom. It appears gradually, first in one muscle, slowly increasing in intensity and spreading to others. Sometimes this spasm is preceded or accompanied by slight sensory disturbance, as pain or sensation of stiffness. It is generally aggravated by emotional influences, if, indeed, it is not present only at such times. The muscles most commonly involved are the sterno-mastoid and trapezius, and, next to these, the splenius capitis. It is an important thing, from a therapeutic standpoint, to determine the muscle in which the spasm commenced, as sometimes the removal of the spasm from that muscle by surgical measures stops it in all secondarily affected. As the spasm extends, the muscles of the face and arm become involved. The abnormal movements always occasion fatigue and discomfort, but rarely absolute pain. Sometimes numbness and tingling in the arm are associated.

(*l*) **Rhythmic or Functional Spasm.**—Under this head may be included a variety of cases characterized by rhythmic movements, and not capable of being classed under any of the foregoing heads. The pathology of all of these cases is unknown, and apparently unknowable. The few pathological investigations seem to demonstrate that they occur independently of organic changes.

2. **Choreic movements,** so named because observed in their most characteristic form in chorea, are characterized by involuntary irregular muscular contractions of single muscles or of muscles in groups, the resulting movements resembling to a certain extent those voluntarily performed. Choreic movements are observed in

a. Chorea.
b. Hysteria.
c. Organic cerebro-spinal disease.
d. Reflex irritation.

(*a*) **Chorea.**—The affection to which the term "chorea" should be applied exclusively is one occurring for the most part in children, characterized by irregular involuntary movements of the voluntary muscles, said movements occurring both during rest and attempts at motion, interfering with the accuracy of voluntary movements, and not infrequently attended by apparent mental weakness. It is closely related to unstable nervous organization, being observed more frequently among girls than boys, and those of neurotic inheritance. Emotional factors play an important part in its etiology, as do also those exposing the nervous system to great wear and tear. It is but seldom due to reflex irritation, though exceptionally one observes cases apparently originating in this way. The relationship between chorea and rheumatism is undoubtedly a close one, but it is hardly to be considered as one of cause and effect, but rather, as I believe, the constitution which predisposes to the one affection also predisposes to the other.

The onset of chorea may be either sudden or gradual. In the former case the child is noticed to begin to twitch, or, more properly speaking, to indulge in restless movements, which rapidly increase in severity and distribution until they are of the most violent character and involve the entire body. When, on the other hand, the disease is of gradual onset, the usual history is that of a slowly-increasing awkwardness or restlessness, for which the child is at times reprimanded. Still another class of cases has the inception of the chorea marked by an apparent loss of power of one or more extremities. Sometimes the parents say that one lateral half of the body is losing strength, and they even express fears of an oncoming paralysis. In extreme cases of this type the loss of power may be general, and so complete as to amount to absolute helplessness. Even in these so-called paralytic cases, careful investigation shows the characteristic restlessness present to some extent. Sooner or later the choreic movements appear, often being severe in direct proportion to the pre-existing paretic condition. The choreic movements themselves need a certain amount of description. In a general way, they may be described by one word: "Restlessness." The child is sitting before one, and it will be noticed to lift one arm, and then drop it again, or it will perform supination and pronation; or it shrugs one or both shoulders; twists around in its chair; shuffles its feet; twists the head; makes grimaces in great variety; or these movements may exist together or follow one another with almost kaleidoscopic variety. In mild cases but few of them are to be noted, and these are of slight degree. In the severer ones they are all present, making their victim a pitiable object, reducing her, as they do, to a condition of absolute helplessness. In extreme cases the muscles of deglutition and respiration are affected. Thus nutrition is impaired, while the interference with breathing causes additional discomfort. The respiratory complication may lead even to irregularity in action of the cardiac muscles, though I am inclined to the view that the latter are not infrequently affected *per se.*

Very important are the heart symptoms associated with this affection. It is especially common to observe a cardiac murmur in the younger patients, this murmur being nearly always a mitral systolic murmur. It practically always disappears when the patient recovers from the chorea. Very exceptionally, it may persist as an organic heart-affection, ultimately leading to most disastrous results.

Of the affections which may justly, by reason of their clinical phenomena, be classed as chorea, the following may be mentioned:

i. Huntington's chorea.
ii. Senile chorea.
iii. Post-hemiplegic chorea.
iv. Chorea of internal inflammations.

(i) **Huntington's Chorea; Hereditary Chorea.**—This affection was first systematically described by Huntington, a Long Island physician, in 1872. It had been previously described in 1841 by Waters, of Franklin, N. Y., in a letter to Dunglison. The most remarkable feature in the etiology of Huntington's chorea is heredity. It has been known to affect four generations, and as many as nineteen subjects in a single family. If one generation escapes, the line of heredity is destroyed. It does not begin until adult life is reached, generally between the ages of thirty and fifty. The exciting causes of the ordinary type of chorea seem to be productive of this disease at times, though, more frequently than otherwise, heredity remains the sole etiological factor. The choreiform movements begin gradually in the upper half of the body, and slowly increase in distribution and intensity. They resemble in character the movements of the chorea of childhood. The eyeballs are often affected, and the mental condition is very frequently considerably impaired. The disease runs a very chronic course of from ten to twenty years. It is essentially incurable. Cases die apparently by reason of the associated mental degeneration. The pathology of the affection is unknown.

(ii) **Senile Chorea.**—Choreic movements sometimes attack people of advanced life, even up to the age of eighty years. Cases of this character are very obstinate to treatment, and generally continue until death takes place from other causes. Depressing emotions and the neurotic constitution seem to play the most important part in their causation. The movements are especially liable to affect the upper part of the body, the involvement of the face and tongue being so severe as to interfere seriously with articulation. The presence of strangers or unusual excitement intensifies the movements. The patient is quiet during sleep. The mind remains unaffected. The general health is not impaired by the disorderly movements. Some few cases recover.

(iii) **Post-Hemiplegic Chorea.**—This must not be confounded with the so-called **paralytic chorea,** which is but the ordinary chorea of childhood in which the paralytic phenomena predominate. Post-hemiplegic or post-paralytic chorea follows attacks of hemiplegia, in which there has been considerable restoration of motor power. The abnormal movements are limited to the affected extremities, and simulate those of typical chorea quite closely. In the vast majority of cases it is associated with hemianæsthesia, especially in its early stages. The commonly accepted view concerning the causation of this condition is that it is dependent upon a lesion of the optic thalamus or of the internal capsule. Sometimes there is associated an incoördination varying from slight instability to pronounced ataxia. It should be noted that while most cases of post-hemiplegic chorea are dependent upon a lesion of the optic thalamus, this condition may also originate in disease of the cortex, crus, pons, and other portions of the pyramidal tracts.

Athetosis, or **mobile spasm,** is a condition closely allied to, if not identical with, post-hemiplegic chorea. It is characterized by involuntary movements, principally in the fingers, and consists of slow movements of flexion and extension, adduction and abduction, in irregular combination and sequence. Athetosis is especially liable to appear in the spastic hemiplegias and diplegias of infancy.

(iv) **Chorea in Internal Inflammation.**—Wood is the only authority, so far as I know, who describes chorea of this type. The movements are said to consist of violent, brusque, widespread muscular contractions, and occur in association with acute inflammatory diseases, as pleurisy, bronchitis, or pneumonia. He remarks that the movements in some of the cases are reflex.

(*b*) **Choreic Movements of Hysteria.**—The sole manifestations of some cases of hysteria are found in certain involuntary or restless movements, which are hard to describe, but which, once seen, exhibit remarkable differences from the characteristics of the involuntary movements observed in chorea. As a rule, however, one depends for his diagnosis not upon the nature of the muscular contractions, but upon associated phenomena or conditions, such as the presence of hysterical stigmata, the age of the patient, etc. In many instances the course of the disease is an important diagnostic criterion, the abnormal movements exhibiting the wonderful fickleness characteristic of hysteria. Diagnoses based upon the association of hysterical stigmata are not always reliable, however, for both hysteria and chorea are not unlikely to occur in subjects having a neurotic inheritance, and the existence of the former affection or temperament is by no means proof that the latter will not enter into the case as a complicating factor. For accuracy in recognizing choreic movements dependent upon hysteria some experience is necessary. Even then it is by no means always possible to reach a correct conclusion until the case has been under observation for some time.

But there are other cases in which the hysterical origin of the movements is evident from their inception. They may be localized to one part or one side of the body, and they are characterized by the association of a persistent rigidity of the affected muscles. Not infrequently they appear as rhythmic spasms recurring at more or less regular intervals. They may be simple, consisting of a definite localized oft-repeated movement, or complex, exhibiting an indescribable sequence of spasms, which may be suggestive of almost any of the spasmodic neuroses.

Of unquestionable hysterical origin is the so-called

Chorea Major.—This affection has likewise no clinical or pathological relation to the ordinary chorea of childhood. Inasmuch as the common name applied to chorea by the laity (St. Vitus's dance) originated in chorea major, it has been customary for authors to consider it in this connection. Chorea major is really a form of religious hysteria, characterized by wild dances and great excitement. For the cure of this, it was customary in the middle ages to make pilgrimages, especially to the shrine of St. Vitus. Epidemics of this character are now seldom observed, although they have occasionally occurred in this century, even in the United States.

Electrical chorea, though having no pathological association with hysteria, as evidenced by its clinical course, may best be mentioned in the present connection. It is a very rare affection, seen almost exclusively in Lombardy and neighboring parts of Italy. It is characterized by spasmodic movements, which consist of sharp, quick, muscular contractions similar to those produced by electrical excitation with the interrupted galvanic current. These movements commence in one extremity, usually the arm, spread to the corresponding leg, and next invade the other side of the body. They increase gradually in severity. Finally the affected limbs become weak, and their muscles atrophy. General paralysis finally supervenes. Then the patient dies. But little is known of this strange affection. Pathological investigations have been very unsatisfactory. Its causes are also unknown. Fright and malaria have been assigned as origins. It affects both sexes and all ages. It has also been called "*Dubini's disease.*"

(*c*) **Choreic Movements of Organic Cerebro-Spinal Disease.**—Choreic movements dependent upon organic cerebro-spinal disease probably have the same pathological origin as the post-hemiplegic variety and athetosis, already described.

(*d*) **Reflex Irritation as a Cause of Choreic Movements.**—In proportion to the actual number of cases of genuine chorea, those originating in some reflex irritation are of rare occurrence; nevertheless, the brilliant results ensuing upon the removal of the exciting cause has created an effect upon the minds of physicians disproportionate to their importance. True it is that the failure to recognize one such case will almost certainly bring discredit upon the physician who fails to discover it. But, on the other hand, we have the damage done to patients by innumerable operations of questionable value for the removal of purely hypothetical irritation. There can be no doubt that in patients with chorea, local irritation of any kind may readily maintain, and even aggravate, the existing nervous irritability. No one will contradict this statement. It follows, then, that it is the physician's duty to examine his patients thoroughly, and direct his efforts to the cure of any and all morbid conditions from which the patient may be suffering. Especial attention should be paid to the eyes, the post-nasal space, the gastro-intestinal tract, and the urinary organs. At the same time the fascination of investigating objective conditions should not lead to neglect of a congenitally unstable nervous organization, and the institution of proper measures for its improvement.

Tremor.

Tremor consists of involuntary oscillatory movements, which are produced by more or less rhythmical alternate contractions and relaxations of antagonistic muscles. It is to be distinguished from fibrillary contractions, a condition characterized by alternate contractions and relaxations of individual bundles of muscular fibres, manifested, not by gross movements of the parts to which the affected muscles are attached, but by wavy subcutaneous oscillations of the fibres involved. Nor should tremor be confounded with muscular twitchings, the muscular contractions in the latter being more decided and less persistent. Clonic movements arising from tension on tendons in cases in which the deep reflexes are greatly in excess have been erroneously denominated "tremor" by some; many of the so-called tremors in spinal and cerebral syphilis and after hemiplegia are undoubtedly of this class.

There are certain characteristics in every case of tremor that should be carefully noted. They are: 1. The amplitude of the tremor, and whether the movements are fine or coarse. 2. The rapidity and regularity of the movements. 3. The regularity of the tremor and the influence exerted upon it by certain external agencies, as rest, voluntary movements, sleep, massage, etc. 4. Its distribution, for it may be limited to single parts or limbs, or to one lateral half of the body; or, again, it may be of universal distribution.

There are times in which patients having tremor do not make that symptom very evident at the time of consulting the physician. A very good test, in order to bring it out, is that of having the patient hold out one hand with the fingers spread pretty wide apart. In patients who are suspected of being in the initial stages of general paresis in which the tremor begins in the lips and tongue, the most satisfactory test is to have the mouth held partly open, with the lips fairly well separated, but thoroughly relaxed.

Tremor may occur apart from diseased conditions. In other words it may be physiological; or at least, under certain circumstances, it may be normal to the individual. Thus it may occur in neurotic subjects under the influence of some violent emotion, as fear, grief, or joy; it may also be produced as the result of severe and rather sustained exertion. In all of these cases it is but temporary, passing away in a short time.

It has been customary for years, in treating of the subject of tremor, to divide cases of it into two great classes, one, *tremor coactus*, in which the movements persist during repose; and the other, *tremor a debilitate*, in which they are observed only during efforts at voluntary movement. In my opinion this classification is not as valuable, clinically, as we have been led to believe, for many of the cases of tremor assigned to the former class have the characteristics of the latter, and *vice versâ*. Regarding the different influences which ameliorate tremulous movements, there is one that I do not find mentioned at all in any literature at my disposal. I refer to massage. Several cases of the most aggravated character have shown a cessation of the tremor for several seconds following manipulation of the muscles involved, or pressure upon certain portions of the trembling extremity. None of these cases was hysterical.

Tremors persisting during rest are typified in paralysis agitans; in fact, this disease is almost the only one in which the movements cease during voluntary movements. Tremors arising from senility, toxic influences, asthenia, hysteria, disseminated sclerosis, and organic disease of the brain, are mostly observed during attempts at voluntary motion. Cases of paroxysmal and epileptic tremor cannot be assigned to either class. They are distinguished from all others in that they occur under such conditions as to make the execution of voluntary movements impossible.

I shall treat of the various forms of tremors under the following heads:

1. Simple tremor, including the so-called essential tremor, and cases arising from hereditary transmission.

2. Senile tremor.
3. Asthenic tremor.
4. Hysterical tremor.
5. Toxic tremor, which may arise from exposure to the following poisons:
    - a. Lead.
    - b. Tobacco.
    - c. Mercury.
    - d. Alcohol.
    - e. Chloral.
    - f. Opium.
    - g. Tea, coffee, etc.
    - h. Uræmia.
6. Tremor symptomatic of or associated with the following-named diseases:
    - a. Exophthalmic goitre.
    - b. Writers' cramp.
    - c. Epilepsy.
    - d. General paralysis of the insane.
    - e. Disseminated sclerosis.
    - f. Paralysis agitans.
    - g. Tumor of the brain.
    - h. Spinal-cord degenerations.
7. Post-paralytic tremor.
8. Paroxysmal tremor.

The occurrence of tremor in the course of cases of multiple neuritis does not prove that this symptom is the result of that pathological condition. As a matter of fact, there is no reason whatever for the production of tremor by nerve-inflammation. But it so happens that the principal causes of multiple neuritis are alcoholic excesses, the toxæmia of the infectious diseases, lead, and arsenic; and it is to these that the symptom is due.

In the above classification no mention has been made of acute or transient tremors, such as those arising in the so-called healthy as a result of physical or mental strain, exposure to cold, etc. The nature of tremors arising from these causes is readily recognized by the merest tyro, and their consideration is unnecessary because they are of so short duration as to rarely, if ever, be brought to the attention of physicians.

Simple tremor includes all those cases in which the tremor occurs without apparent cause, or is unassociated with other evidences of ill-health. In many instances it has shown a remarkable tendency to occur in families; so much so, in fact, as to lead some authors to establish a separate class, called hereditary tremor. In all these cases the tremulous movements are very fine, and generally regular as to their amplitude and rhythm. They are for a time under the control of the will. While intensified during voluntary movements, they do not interfere in the least with the execution of the same. The patient can perform such complex movements as writing without difficulty. Though not necessarily so, the tremor is limited to the hands and head. It affects mostly the young and middle-aged. It is very intractable to treatment. Once it has thoroughly established itself, it persists throughout life. Some tremors occurring in children born of nervous mothers disappear spontaneously, or under treatment after a few years.

The hereditary tendency to tremor displayed by some families is truly remarkable. Thus, in a case reported by Dr. Samuel West, the patient's grandmother and mother had been affected. He was one of ten children, all of whom suffered from tremor varying in degree, and the affliction had been reproduced in his own six children.

Senile tremor appears in people of advanced years and in the prematurely aged. It presents points of similarity to each of several varieties of tremor. It is a fine tremor, aggravated by movement, beginning usually in the head and neck, the arms and hands being attacked after the trouble has lasted some time. Clinically, it is especially apt to be confounded with paralysis agitans. In the latter the tremor is present when the hands are at rest; the movements usually begin in the hands, while the head and neck are generally free, and the tremulous parts are affected with a peculiar rigidity.

Senile tremor is nearly always bilateral from the beginning; in paralysis agitans, one part, usually the hand, is first attacked, whence it extends slowly to the rest of the body. In the beginning of senile tremor there is aggravation during voluntary movements, if, indeed, it exists only at such times. This is very different from the condition obtaining in paralysis agitans.

Asthenic tremor has all the characteristics of the simple variety, but differs from it in having a well-defined relation to certain asthenic conditions. It is especially liable to occur after exhausting diseases, as the specific fevers, in cases of nervous exhaustion, and after hæmorrhages and sexual excesses. Many of the cases of tremor in young males arise from the latter cause. The tremulous movements occurring during the course of diseases other than of the nervous system are of the asthenic variety. They are significant only of the extreme debility present.

Hysterical tremors are exceedingly irregular in every respect. They may be fine or coarse, rapid or slow, mild or severe, and these in the one patient. Their distribution is as varied as their character. They may be hemiplegic, or they may invade the entire body, or they may be limited to but one limb or to one group of muscles. The movements are intensified during the execution of voluntary movements, but, as a rule, the aggravation does not begin immediately after the movements are commenced, usually not asserting itself until the muscular action has been maintained for some little time. Hysterical tremor is almost always increased when attention is directed to it, and ceases when the mind is diverted. It may in some cases be associated with paralysis and contractures. When the limbs are the seat of contracture, the tremor is excited by any attempt to move them passively, or even by the manipulation necessary during a medical examination. A long duration (*e.g.*, many months) does not contra-indicate the hysterical nature of a given tremor. Dutil truly described hysterical tremors in few words when he designated them as polymorphous.

Toxic Tremor.—As already stated, chronic tremors may be produced by exposure to certain poisons, as lead, mercury, tobacco, alcohol, tea, coffee, chloral, opium, etc. There is little to say that is diagnostic concerning tremors arising from exposure to these poisons. They are all fine tremors, usually intensified during voluntary movements. *Saturnine tremor* is usually limited to the face and hands. Occasionally it invades the muscles of the face, neck and tongue. It is aggravated by emotions and after prolonged exercise, or in the latter part of the day, when the muscular system is exhausted. It occurs almost exclusively in chronic cases of lead-poisoning. The diagnosis depends upon a history of exposure to lead, the previous existence of colic, blue line on the gums, anæmia, wrist-drop, etc.

Tremor is of far more frequent occurrence in cases of poisoning by *mercury* than by the metal above mentioned. It usually begins in the face and tongue, and is always intensified by any motion. In its incipient stages it occurs only during voluntary movements, but later it becomes constant, in which case it presents a remarkable similarity to paralysis agitans. Articulation is very much interfered with. As a rule, the tremor ceases during sleep; but there are very notable exceptions to this. Its occurrence is favored by addiction to alcohol and tobacco. In some cases the tremor is of sudden onset. The diagnosis rests upon the known exposure to fumes of mercury, and upon the associated symptoms. A very interesting as well as rather unusual case of mercurial tremor has been reported by Letulle. A man, æt. 39, had been exposed to the fumes of mercury for many years. In his first attack of tremors the movements were worse in the morning, and much relieved between the hours of 4 and 5 P.M., and again very marked in the evening. One day the tremors suddenly became very violent,—so much so, in fact, that the man was thrown down by them and convulsed so that he could not walk. When perfectly at rest in bed the movements were but slight, but they were very much intensified by voluntary effort. Motions were extensive, rapid and arrhythmical, and worse on the left side. An anæsthetic area was discovered on the back of the right forearm and hand. Vision had become impaired. Constricting the limb lessened the tremor. Finally the tremor was stopped permanently by ligaturing the limb and passes with a magnet. The author called it a case of mercurial hysteria.

Acute and chronic alcoholism are almost always attended by tremor of some kind, noticeable usually in the arms, face and tongue, often associated with local muscular twitchings. In severe cases the latter amount almost to shock-like muscular contractions. The alcoholic tremor is especially apt to be worse in the morning, and is promptly relieved by a drink of whiskey. The restlessness of alcoholic subjects makes the tremor very prominent.

Tremor is not a common symptom of the *chloral habit.* When it occurs as a result of addiction to chloral or opium, it resembles in every way that arising from alcohol.

The tremor caused by excessive use of tobacco, coffee, tea, etc., is of very fine character, apparently presenting no distinctions from simple tremor.

Charcot has described a form of tremor closely resembling that of paralysis agitans, due to uræmia.

Tremor is met with as a prominent symptom in quite a variety of diseases. In **exophthalmic goitre** it is rarely absent. It is generally not of a severe grade, usually being mentioned by the patient as a nervousness, and observed by the physician as an exceedingly fine tremulous movement. Still it may be of variable character. Sometimes it is so violent and regular as to resemble that of paralysis agitans. It may be general, or, again, confined to the hands alone, or even accompanied by fibrillary contraction of the muscles of the face. This fact must be borne in mind lest a diagnosis of general paralysis be too hastily made, especially as exophthalmic goitre is not infrequently associated with mental symptoms. The usual mental anomaly, however, is melancholia, or melancholia alternating with periods of considerable excitement. It has been known to be unilateral in distribution, when the exophthalmos or the goitre was one-sided.

Tremor is occasionally met with as the almost exclusive manifestation of **writers' cramp.** It is then most marked in the forefinger when the hand is at rest and the fingers slightly separated. It is especially apt to be produced by prolonged work and over-fatigue.

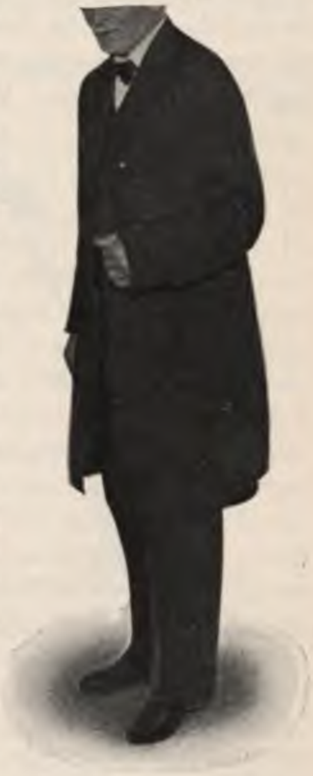
Rare cases of **epilepsy** are met with in which the seizures are preceded by a general or localized tremor as a kind of aura, or, as in one of my patients, tremor took the place of the convulsions. Gowers holds that "fits" ushered in by tremor are almost always epileptic, and not hysterical.

General paralysis of the insane in its earliest stages presents more or less tremor. The movements are especially marked in the tongue and lips, less so in the fingers. Speech becomes hesitating and drawling, with a tendency to slur words. Its relation to the severity of other symptoms is decidedly irregular. In the final stages of the disease, violent tremor accompanies every voluntary movement.

Disseminated sclerosis and **paralysis agitans** are, of all diseases, the most constantly associated with tremor; in fact we might call tremor a necessary part of their symptomatology. These diseases have a well-defined clinical course.

Paralysis agitans is a disease of obscure etiology and uncertain pathology, characterized by tremor in association with more or less general rigidity, muscular weakness, and peculiar gait and posture. It is a matter of observation that nearly all cases occur after the age of fifty, and that it seldom attacks subjects before the age of forty. The prevalent view concerning its nature makes it a premature senility. In the vast majority of cases paralysis agitans is of insidious onset. Sometimes, however, under the influence of a sudden shock, symptoms have made their appearance with the rapidity observed in apoplectic seizures. The first symptom observed is usually a tremor of mild degree, gradually increasing in severity over a term of years. Almost always the tremor begins in one or the other hand. It then spreads to the leg on the corresponding side; then, crossing to the other side, involves the arm and leg in order. It may exceptionally invade both hands and extend to the legs, or it may involve the arm of one side and the leg of the other; or, again, both legs only may be affected. In the early stages of the disease the tremor is observed only when the affected part is at rest. Attempts at voluntary movement control the movements completely. Later, however, motion exerts no influence whatever on them; in fact it may have the reverse effect, that of greatly aggravating them. The tremulous movements as involving the upper extremity may be described as follows: The hand is held in the writing position, the tremulous movements in the fingers, consisting of metacarpo-phalangeal flexion and extension, bearing similarity to the movements employed in spinning wool (Charcot) or rolling a pill. There are also rhythmical flexion and extension at the wrist, and supination and pronation at the forearm. The arm is flexed at the elbow. The tremor ceases entirely during sleep.

FIG. 193.



Case of paralysis agitans.

Muscular weakness is a constant symptom, though not a prominent

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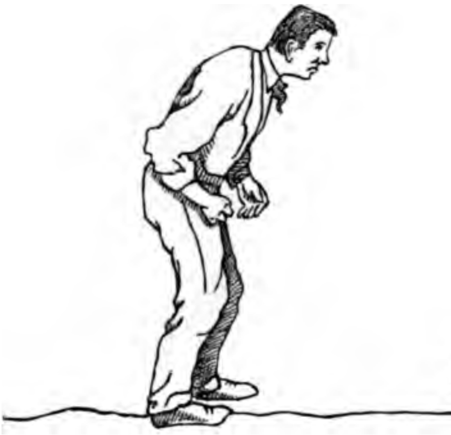
one. Occasionally it may exist to a marked degree, under which circumstances the tremor is proportionately severe. Still more rarely, the weakness may be great and the tremor entirely absent. The conclusion that a slowly progressive hemiplegia is at the bottom of the trouble seems almost irresistible. The muscular weakness in paralysis agitans never amounts to complete loss of power.

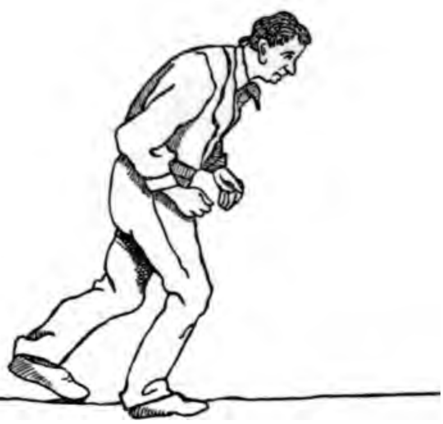
The rigidity and the peculiar attitudes of the patient are as characteristic of the disease as is the tremor. The muscles everywhere are rigid, this condition being especially marked in the flexors. The back is bowed; the head is flexed on the chest. The arms are held flexed at the elbow and slightly away from the body. The hands are in the writing position, and drawn slightly towards the ulnar side of the forearm. The legs likewise show involvement. With the patient standing, there is slight flexion at both hips and knees. The muscular rigidities and contractures are the cause of the characteristic gait. The patient moves as if he was soldered together, so to speak, at his joints. In starting to move he manifests considerable difficulty, but having once begun, he proceeds fairly well. He may even show a tendency to run, taking quick, short, shuffling steps. The body being bent forward with this gait has led to its being described "as if the patient was pursuing his own centre of gravity." Charcot has called this phenomenon in walking "propulsion." A very slight push will cause the patient to pitch forward. A slight pull backward will cause him to fall in that direction, this being called "retropulsion." In some few cases a lateral pull will produce "lateropulsion." Charcot believes these phenomena partake of the nature of forced movements, though Strümpell, with apparently good reason, thinks that they arise from the displacement of the

FIG. 194.



Attitude (exaggerated) of paralysis agitans.

FIG. 195.



Paralysis agitans. Festinating gait (exaggerated).

—After Dana.

patient's centre of gravity by his posture. The muscles of the face exhibit their involvement by their immobile expression. The trunk seems to be more affected than the extremities. It is said that the muscles of the eyes are rarely implicated.

The speech exhibits several abnormalities. The most constant is a peculiar monotonous, expressionless voice. Another, first described by Buzzard, consists of a shrill, piping tone, comparable with that assumed by actors in their stage impersonations of aged men. And still another is the vocal counterpart of the gait, if the reader will permit the expression. The patient finds considerable difficulty in starting his speech, but, having once begun, proceeds with comparative fluency.

Disseminated sclerosis of the brain and spinal cord, being dependent upon pathological changes scattered in foci of different sizes throughout the central nervous system, must present the greatest possible variety of symptoms, depending upon the functions of the particular structures involved. So great is this variety that one may well be justified in asserting—in declaring—that disseminated sclerosis of the brain and cord has no characteristic symptomatology. The symptoms include paralysis, tremor, ocular phenomena, and disturbances of speech. The paralytic phenomena will vary according to the particular portion or portions of the motor tract affected. The tremor is the symptom upon which, above all others, most emphasis has been made. It consists of irregular movements of a part during efforts at voluntary motion. The amplitude of these movements is large, and they are never rhythmical, as in paralysis agitans. They cease absolutely while the patient is at rest. I feel that they are better characterized as jerking rather than as tremulous in character. The ataxia resembles that of tabes, but the association of symptoms enables one to make a ready differentiation from that disease. The eye-symptoms consist of nystagmus, amblyopia, and diplopia. The disturbances in articulation are highly characteristic, when present. These consist of what has been denominated "scanning speech," or "syllabic utterance." There is apparently an ataxia or jerkiness of the muscles of articulation, and this leads to slow and studied utterance on the part of the patient to insure his being understood. The patient may present almost any type of mental change. Usually the intellect is dulled and the memory weak. The knee-jerks are usually exaggerated, and ankle-clonus is present.

Some cases of **brain tumor** present tremor as a prominent system. Usually, it has but little significance. Sometimes, however, pressure of a cerebellar tumor upon the pons varolii or medulla oblongata, or of a tumor of the centrum ovale upon the fibres of the internal capsule, causes a persistent tremor. In a case of cerebellar tumor (sarcoma of the dura mater) under my care, and in which there was marked irritation of the pons, there were repeated paroxysms of tremor, limited to the arm on the side opposite to the lesion. The attacks lasted all the way from a few hours to a day or more.

Following paralytic seizures, we sometimes have a tremor affecting the paralyzed extremities. This varies in different cases. Sometimes its character is such as to lead to its being called post-paralytic chorea.

Paroxysmal tremors have been described by different authors. Suckling reports the case of a laborer, aged sixty years, who for two years had suffered from tremor of the right upper extremity. Attacks were induced by sudden noises or shocks. The movements were limited to the right upper extremity only. They were of great amplitude, and consisted of very rapid flexion and extension of the forearm, which could not be controlled voluntarily or by force. There was no aura preceding the attack, but coincidently with the onset of the tremor the patient had pain in the upper part of the right thigh. There was no loss of power in the right arm or leg, and no alteration in the reflexes. The attacks ceased abruptly and spontaneously—as the patient said, with a snap. Dr. Suckling expressed the opinion that the tremor would eventually become continuous, and the case end in paralysis agitans.

The **convulsive tremor** of Hammond, described by him in 1867 (and in his work on *Nervous Diseases*, 1886) as including those cases of non-rhythmical tremor or clonic convulsive seizures which are unattended with loss of consciousness, but which, nevertheless, are paroxysmal in character, in the light of modern neurology requires no consideration. Such cases may all, or nearly all of them, be relegated to various forms of tolerably well-understood nervous conditions, without the necessity of manufacturing a new disease.

As to the possibility of simulation of tremor, I believe that it can be done, and think that I have seen one such case,—one in which the movements were limited to one arm, and which ceased on the recovery of substantial damages.

Paramyoclonus multiplex is probably better included under the tremors than elsewhere. It is an exceedingly rare disease, not more than a dozen cases of the same having been reported. It is characterized by paroxysmal clonic movements, symmetrically distributed in muscles, or groups of muscles, of which one extremity has an attachment to the trunk. The muscles of the forearms, wrists and hands, and legs and feet, are not affected. The majority of cases occur in males. The prognosis is favorable, although the disease runs a slow course.

Contractures and Rigidities.—For clinical study it is well to make a distinction between contracture and rigidity. The former term should be restricted to indicate persistent rigidity or contraction of muscle, such a condition being dependent upon local structural change, so that the affected muscle cannot be extended to its normal, under anæsthesia, without rupture or section. The term "rigidity" should be limited to cases in which local structural changes have not taken place; hence the parts may be manipulated into their normal positions. In some cases the forcible reduction of a rigidity may occasion rupture of muscular fibres, unless the parts are replaced under an anæsthetic. It is essential to remember that a muscle or part which has been in a state of rigidity for a long time ultimately undergoes organic changes, and thus the rigidity is replaced by contracture.

1. **Rigidities** may occur in
   - a. Cerebral affections.
   - b. Spinal affections.
   - c. Diseases and lesions of the peripheral nerves.
   - d. Functional nervous disorders.
   - e. Muscular disorders.
   - f. Joint lesions.

(*a*) **Rigidity of Cerebral Affections.**—The cerebral affections which may exhibit rigidity at some time in their clinical courses include the following:

- i. Cerebral hæmorrhage.
- ii. The different varieties of meningitis.
- iii. Cerebellar disease.
- iv. Tumor of brain.
- v. Cerebral thrombosis.
- iv. Cerebral embolism.

The rigidities ensuing upon the acute cerebral lesions, *i.e.*, hæmorrhage, embolism and thrombosis, may be classified according to the period at which they appear as immediate, early and late rigidity. Each of these is due to definite pathological conditions, and possesses diagnostic significance.

Immediate rigidity appears with the onset of the lesion, and is due to the direct irritating effects of the same upon the pyramidal tracts. It is limited to the paralyzed parts. As a rule, it is not sufficiently marked to be observed unless looked for. It will then be recognized by some slight resistance to passive motion of the paralyzed parts. It usually disappears during sleep.

Early rigidity is the term used to describe a rigidity which appears in the paralyzed parts in from two to four weeks after the appearance of the lesion. It likewise is due to irritation of the pyramidal tracts, but in this case inflammation is the exciting cause. Immediate and early rigidity possess an unfavorable prognostic significance.

Late rigidity sets in at any time after one or two months from the onset of the lesion, and is dependent upon degeneration of the pyramidal tracts. While the deep reflexes are exaggerated in all cases of hemiplegia, they are especially so in those associated with late rigidity. The affected muscles ultimately undergo atrophy, and the long maintenance of the rigidity ends in their contracture. The usual position of the arm in these cases is that of flexion to a right angle at the elbow, with exaggerated flexion of the wrist and fingers. The rigidity of the fingers may be so great as to threaten the structural integrity of the tissues of the palm of the hand. The position of the leg is that of flexion at the knee and ankle.

The **rigidity of cerebellar disease** is rather an uncertain quantity. Undoubtedly, in many instances, there is rigidity with backward deviation of the head, due in all probability to meningeal irritation. As to other muscular rigidity than the above, the most diverse possible opinions prevail. Some contend for rigidity, others deny its presence; some claim that the rigidity is unilateral, others that it is bilateral. It will thus be seen that this symptom, though probably present in many cases of cerebellar disease, is so inconstant as to be devoid of diagnostic value. When present, it is undoubtedly dependent upon lesions of structures adjacent to the cerebellum.

In the different varieties of **meningitis**, rigidity is not an uncommon symptom. It is not, however, an important one from a diagnostic standpoint, excepting in the epidemic cerebro-spinal variety, in which disease it is always present to some degree at least. The rigidity in the latter affection is to be sought, in mild cases, in the back of the neck, and is exhibited by a sense of resistance on attempting to flex the head on the chest. In the more severe examples of the disease, the rigidity may cause most remarkable retraction of the head, even to the extent of interfering mechanically with deglutition.

Kernig's sign may be classed as a symptomatic evidence of rigidity, and is a valuable diagnostic symptom. It is not pathognomonic, however, as it is occasionally absent in meningitis, and is sometimes present in other diseases. To elicit this sign, the patient is placed upon his back. The thigh is flexed at a right angle upon the abdomen. Under normal conditions it is possible, with the body in this position, to extend the leg at the knee on a line with the thigh. But in meningitis this complete extension at the knee is nearly always impossible. Very often, indeed, it cannot be extended at a greater angle than 90 degrees, and never beyond 135.

(*b*) **Rigidity of Spinal-Cord Affections.**—The spinal-cord affections which may exhibit rigidity as one of their symptoms include in a general way those presenting meningeal inflammation, and those depending in whole or in part upon degeneration or other lesion of the pyramidal tracts. Specifically, they are as follows:

i. Acute myelitis.
ii. Acute meningitis.
iii. Chronic meningitis.

iv. Scrofulous pachymeningitis from vertebral caries.
v. Meningeal hæmorrhage.
vi. Spastic paraplegia.
vii. Amyotrophic lateral sclerosis.
viii. Bulbar paralysis.
ix. Tumor of spinal cord.
x. Disseminated sclerosis.

In the inflammatory affections of the spinal meninges, rigidity is an almost constant feature. As in the cerebral varieties, it may be present to any conceivable extent from the scarcely perceptible, which may readily escape detection, to the most marked degrees. It may be so severe and persistent as to give the impression that there are organic changes in the muscles; for the affected extremities, usually the lower limbs, cannot be straightened, and the parts may not relax during either sleep or anæsthesia. The rigidity is due to the irritation of the sheaths of the spinal nerve-roots by reason of inflammation propagated from the spinal membranes. Severe pains are associated, and constitute a valuable symptom by which the nature of the rigidity may be differentiated from that of certain cases of hysteria. An additional diagnostic aid is found in the association of more or less extensive paralysis arising from the pressure of the inflammatory exudate.

In acute myelitis, rigidity is not a very prominent symptom. When present, it is dependent upon an extension of the inflammation to the spinal coverings. Peripheral pains and paralyses are far more prominent symptoms in myelitis than they are in meningitis.

The rigidity arising from lesions of the pyramidal tracts is typified in spastic paraplegia. It has not inaptly been described as "*clasp-knife*" *rigidity*. It is especially marked in the extensor muscles, and is greatest when the legs are extended. Taking the rigid member in the hands and endeavoring to flex it at the knee, one finds that flexion to a certain degree is accomplished by the expenditure of quite considerable force. But having attained that point, force is no longer required to produce flexion. Conversely, when producing extension, up to a certain point the aid of the hands is required, and then the limb seems to spring straight of itself. These cases are associated with increase of all the superficial and deep reflexes; ankle-clonus is marked; and the gait is of the characteristic spastic variety.

The rigidity in disseminated sclerosis, bulbar paralysis, spinal-cord tumor, and amyotrophic lateral sclerosis, receives the same explanation as in spastic paraplegia. In bulbar paralysis it is a late phenomenon, and occurs only after the paralysis of the lips, tongue, pharynx and larynx has become complete, and there is beginning involvement of the extremities. In disseminated sclerosis and amyotrophic lateral sclerosis it may appear early or late, depending upon the period of these diseases at which the pyramidal tracts are involved.

(*c*) **Rigidity from Disease or Injury of the Peripheral Nerves.**—It is very exceptionally, indeed, that rigidity may occur as one of the phenomena of neuritis. When present, the diagnosis is based upon the history of the case, and the presence of pain or tenderness along the course of the affected nerves.

(*d*) **Rigidity from Functional Nervous Disease.—Hysterical Rigidity.**—Hysterical rigidities are exhibited by tonic spasm of the affected parts. They may come on suddenly or gradually; after a convulsion or injury, or violent emotion, or spontaneously; and they may last for a short time, or they may continue for months; and when they disappear, they may do so suddenly, without apparent cause. While the rigidity lasts, it continues about the same from day to day; but not infrequently it presents great variations in intensity. It is increased by any attempt to overcome it. It often persists during sleep. The arms are more frequently involved than the legs, the position being one of flexion at the different joints, thus simulating the late rigidity of hemiplegia. In one important particular does it differ from the latter, however; when passive motion is performed at the wrist-joint, the flexion of the fingers is not lessened thereby. Anæsthesia nearly always exists to some degree in almost all cases in the limbs whose functions are disturbed. From long-continued contraction muscles may undergo organic alterations, the shortening becoming permanent.

(*e*) **Rigidity from Muscular Disease.**—Rigidity originating in disease of the muscles finds an example in **Thomsen's disease,** also known as *myotonia congenita.* The onset of the disease is gradual, the difficulty in executing voluntary movements being, as a rule, attributed to awkwardness. As soon as the patient attempts to make a movement the muscles concerned become rigid, and the limb is held motionless. After several seconds the muscles relax and the movement is accomplished, the rigidity reappearing on the attempted execution of the last movement. In some cases the rigidity is limited to the muscles concerned in voluntary action; in others it becomes universal after the long continuance of the disease. It sometimes happens that difficulty is experienced only when beginning the movements; that is to say, the patient finds it difficult to walk immediately on rising from a sitting or lying posture, but meets with little trouble after he once gets started. The difficulty is mostly limited to the muscles of the extremities, although, in rare instances, those of the face and eyeballs become involved. The tonic contractions are always painless. Sensory symptoms are absent. The deep and superficial reflexes are normal. Mental deterioration sometimes takes place. The affected muscles give the appearance of good development and great strength, but in reality they are weak. The tonic contractions are aggravated by cold and mental excitement. They are much relieved by judicious exercise.

(*f*) **Rigidity from Joint-Lesions.**—The rigidity from joint-lesions, usually inflammatory in character, is to be regarded as a conservative symptom, as it is one of nature's methods for securing local fixation and rest. Sometimes, however, it is harmful in that it exerts abnormally great pressure in the apposition of the inflamed joint-surfaces. In all cases it constitutes a very important or delicate means of diagnosing joint-lesion in its incipiency, as shown in the impaired movements in hip-disease and the rigidity in vertebral caries. In the more acute joint-inflammations pain is such a prominent symptom that rigidity loses much of its diagnostic value, though always present.

Contractures or Contractions.—Contractures or contractions, *i.e.*, rigidities in which the contracting muscles have undergone organic changes, and have thereby become fixed in their abnormal positions, may result from the persistent and prolonged continuance of a rigidity, or they may, and frequently do, occur in the late stages of *acute poliomyelitis anterior*. The class of cases first mentioned have been sufficiently elaborated in the preceding paragraphs relating to rigidity and its causes. The contractures in acute poliomyelitis anterior appear in the late stages of the disease, and usually produce one of the varieties of club-foot. The exact mechanism of the deformities thus ensuing has been and is still a matter of debate; hence this subject need not be discussed here. It is sufficient to know that they do occur. It is generally observed that those muscles which have been most severely involved in the paralysis are overpowered by their antagonists, which contract, and finally undergo organic changes, thus fixing them in their abnormal positions. The anterior tibial group of muscles usually being the weakest, it is the calf-muscles that contract, and the resulting deformity is a talipes equinus or equino-varus.

Disturbance of Sensation.

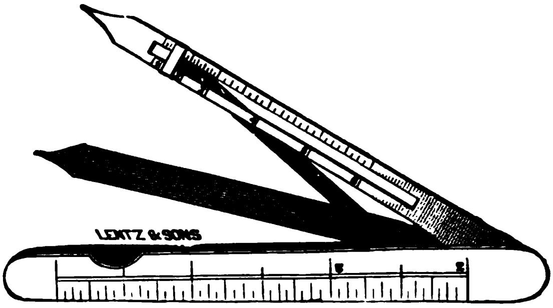
The clinical study of sensory functions involves investigation of the various components of sensation, which it is important to remember need not be involved in equal degree. The various sensory components above referred to include

a. The tactile sense.
b. The temperature sense.
c. The pain sense.
d. The muscular sense.
e. The stereognostic sense.

These, like the motor functions, may be affected in the directions of increase, decrease, and alteration of sensibility, known respectively as hyperæsthesia, anæsthesia, and paræsthesia. As types of paræsthesia, we have transferred sensations also, called allochiria. Delayed sensation may be regarded as a type of anæsthesia.

(*a*) **Examination of the Tactile Sense.**—In the investigation of this, as of other sensory functions, the physician must depend very largely upon the intelligent coöperation of his patient. Under the best of circumstances this is by no means reliable. It is important at the outset that the examination be so conducted that whatever replies are given by the patient are based upon perceptions received through the sense of touch exclusively. Hence the patient should be blindfolded, or in some other way prevented from seeing what is going on. Otherwise vision will be utilized to make up for any deficiency of sensibility. Examinations to determine the condition of sensibility are time-consuming at the best. The one which will appeal to most examiners as being practical is that by drawing a tuft of cotton or a camel's-hair brush over the suspected areas. The patient should be directed to say " Yes " or " Now " when he perceives the touch, and " No " when he does not. The tip of the finger should not be used for making the impressions, because of the probable difference in temperature between the examiner's finger and the skin of the patient. When the anæsthesia is hemilateral, it is well to make comparison of corresponding portions of the two sides of the body.

FIG. 196.



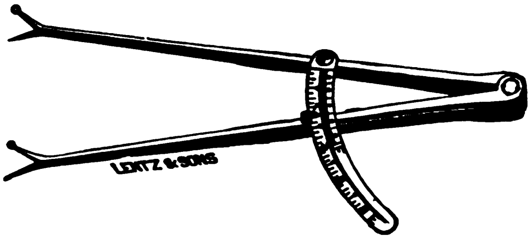
Vance's Æsthesiometer.

The tactile sense may also be investigated by means of an æsthesiometer. This instrument is manufactured in different shapes, the general plan of construction being that of two rather dull points, capable of being moved nearer together or farther apart, at the will of the examiner. The most popular instrument is in the form of a compass. (Fig. 197.) In using it the patient is blindfolded, so that his vision is precluded from making up for any deficient sensibility. The principle on which it is used depends upon the fact that as the acuteness of tactile sensibility increases, the two points can be distinguished as separate points when more closely approximated. Certain portions of the body are normally more sensitive than others, *e.g.*, the tongue and tips of the fingers. Others, as the back and the thighs, possess comparatively poor sensibility. The following table gives the average distances at which the two points can be distinguished:

Tip of tongue	1 mm.
Tip of fingers	2 mm.
Lips	3 mm.
Dorsal surface of fingers	6 mm.
Tip of nose	8 mm.
Forearm	9 mm.
Tip of toes, cheeks, eyelids	12 mm.
Temple	13 mm.
Back of hands	30 mm.
Neck	35 mm.
Forearm, leg, back of foot	40 mm.
Back	60 to 80 mm.
Arm and thigh	80 mm.

In using the æsthesiometer care must be observed that pressure is exerted equally on the two points, and that they be made to touch the skin simultaneously. They are also more readily distinguished as separate points when side by side at right angles to the long axis of the body or limbs.

FIG. 197.



Carroll's Æsthesiometer.

Another very useful form of æsthesiometer is one the arms of which are provided with two branches, these being sharp and blunted respectively. It is to be used in determining the patient's ability to recognize sharp and blunted points.

Rumpf has suggested that **tactile sensibility** be tested by writing on the skin with a hard-pointed instrument, the patient's eyes being closed. The more acute the sensation, the smaller are the characters that may be recognized. On the finger-tips, the healthy subject should be able to recognize letters one-fifth of an inch in height. This method of investigating sensibility is worthy of attention. It impresses me, however, that in order to respond normally to this test, other sensory functions than mere touch must be intact.

To test the **temperature sense,** test-tubes containing hot and cold water may be applied in irregular alternation to the surface of the body, the patient being directed to say whether the one in contact is hot or cold. This test may also be used to determine the delicacy of the temperature-sense, if a little care be taken. Ordinarily, differences of temperature of three degrees Fahrenheit are appreciable within the limits of 32° F. and 104° F.

The **pain sense** may be tested by pricking the skin with a clean needle, or pinching a fold of skin, or by the application of the faradic brush.

Muscular sensibility is determined by the ability of the patient to recognize weights.

The **stereognostic sense** is tested by the ability of the patient to recognize the nature of objects held in the hand,—cubes, discs, spheres, ovoids, coins, etc., being employed as test-objects.

The **sense of locality** is to be determined by the ability of the patient to touch with his finger the part previously touched by the finger or instrument of the examiner. Too much accuracy must not be expected of the patient, for even in health it is not always possible for the patient to specify the exact spot. Oppenheim specifies that a difference of one centimetre on the hands, and from two to four centimetres on the arms and legs, is within the bounds of health.

The **special sensibility of joints** is determined by making passive movements of the limbs, and having the patient state the positions in which the part moved has been placed. Too great variations in the positions of the limbs should not be made. Care must be observed, when making the passive movements, that the patient is not enabled to determine the change in position by the direction in which the pressure has been exerted. Hence, when making the test, the limb should be grasped with a firm circumferential pressure.

Anæsthesia is a term conveying the diminution or loss of any of the sensory functions, the special one involved being used as a qualifying adjective; thus deficient sensitiveness to temperature being referred to as thermo-anæsthesia. When the term is used without qualification, it refers to the tactile sense only. Most of the varieties of anæsthesia have their special terms. **Analgesia** is the loss of the pain sense; **astereognosis** or stereoagnosis indicates loss of the ability to recognize the shape or nature of objects. As to the muscular and articular senses, no special designation has been recommended, it generally being regarded as more practical to speak of them as normal or deficient, as the case may be.

According to its distribution, anæsthesia has received the following designations:

Hemianæsthesia, when the loss of sensation is limited to one lateral half of the body.

Paranæsthesia, when limited to the lower extremities.

Monoanæsthesia, when the loss of sensation is confined to one part of the body, as an arm or a leg.

Alternate hemianæsthesia, when there is anæsthesia of one-half of the face and of the opposite side of the body.

Anæsthesia may also involve certain of the viscera, in which case we are accustomed to designate it **visceral anæsthesia.**

Anæsthesia must always be regarded as dependent upon functional or organic disease of the sensory tract between the cortex of the brain and their termination in the sensory organs in the skin. In organic affections, the lesion must be situated in the peripheral nerves, the spinal cord, the medulla oblongata, the pons, the internal capsule, the sensory projection tracts, or the cortex cerebri.

Anæsthesia dependent upon disease of the peripheral nerves we call **peripheral anæsthesia.** It is limited to an area supplied by some special nerve-trunk or one of its branches. If the nerve be one of mixed motor and sensory functions, there is associated paralysis. The anæsthetic part is in that portion of the body moved by the paralyzed muscles. The exact areas of anæsthesia in lesions of the various peripheral nerves are not exactly the same for all individuals, but only approximately so. Nor are the boundary lines of the same sharply delimited.

The peripheral nervous affections capable of producing anæsthesia are the following:

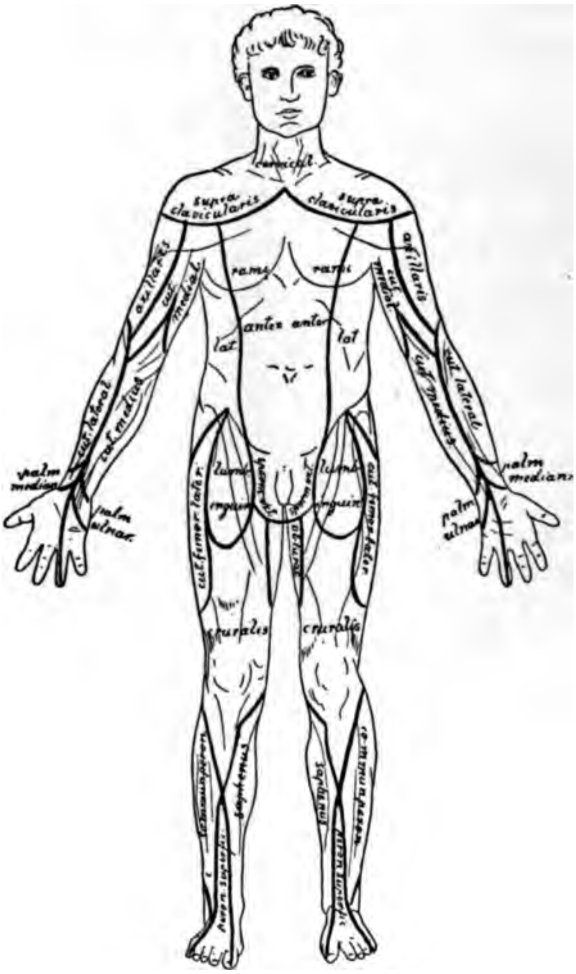
a. Simple neuritis.
b. Multiple neuritis.
    i. Beri-beri.
    ii. Alcoholic.
    iii. Post-diphtheritic.
    iv. Post-typhoid.
    v. Post-influenzal.
    vi. Plumbic.
    vii. Arsenical.
    viii. Malarial.
c. Herpes zoster.

Peripheral anæsthesia may also occur as a symptom of

a. Exposure to cold.
b. Defective circulation.
c. Excessive indulgence in tea.
d. The various acroparæsthesiæ.
e. Leprosy.
f. Changes in the cutaneous structures.

(*a*) **Simple Neuritis.**—The diagnosis of this affection rarely depends upon the recognition of localized anæsthesia simply because the other symptoms, notably the paralysis and the pain, are far more obtrusive, the disturbance of sensation being a matter of secondary consideration, so far as the patient's feelings are concerned. The paralytic phenomena of neuritis in different situations have already been presented (*vide* page 730), and the diagnostic value of pain will be studied very presently. Words are of but little value in describing the anæsthetic areas dependent upon disease or injury of the many spinal and cranial nerves and their branches; hence the reader must rest satisfied with the accompanying illustrations, which demonstrate as accurately as such illustrations can the localizing value of this symptom occurring in diverse situations.

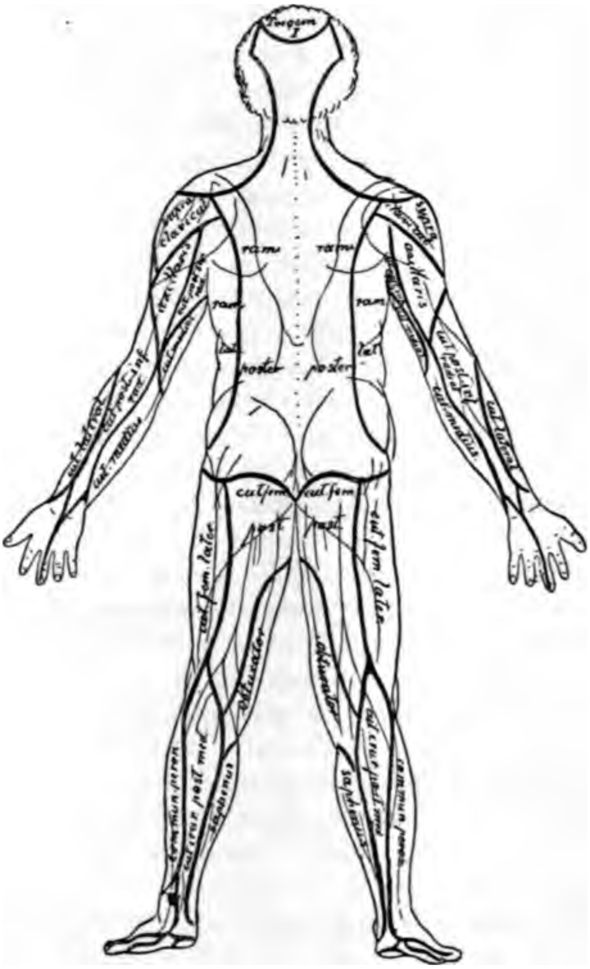
FIG. 198.



Distribution of cutaneous nerves.—After Oppenheimer.

(*b*) **Multiple Neuritis.**—In multiple neuritis the value of anæsthesia as a diagnostic symptom is great. In very many instances it constitutes the first symptom, and, moreover, the one which is the most obtrusive to the patient throughout the course of the disease. This statement applies with especial force to the neuritis following diphtheria, commonly known as post-diphtheritic paralysis. It of itself may be the fundamental factor in causing motor symptoms, *e.g.*, the pseudo-ataxia, which is commonly present. The prominence of anæsthesia in many instances should not lead to the neglect of a search for other phenomena, for these are almost always present, including motor paralysis and loss or diminution of the deep and superficial reflexes. Pain may be present, but it is commonly absent. The localization of the anæsthesia and the palsy is ofttimes characteristic, being noted with especial frequency in the throat.

FIG. 199.



Distribution of cutaneous nerves, posterior surface.—After Oppenheimer.

Post-typhoid neuritis may be either simple or multiple. Pain is commonly a prominent feature, not infrequently being agonizing in character, especially in the localized forms. It may occur either as a complication or as a sequel of the disease. The severity of the primary disorder seems to have no influence in its production. The associated paralysis is of the atrophic variety, and exhibits the reaction of degeneration. A variety of post-typhoid neuritis, known as *tender toes*, may be referred to in this connection. It is not of common occurrence, and was apparently unknown before the introduction of the Brand treatment. The affection is characterized by extreme sensitiveness to pressure, even slight, of the tips and pads of the toes. The dorsum of the foot is never involved. Post-typhoid neuritis may end fatally. It is only exceptionally that the paralysis is permanent.

In **alcoholic neuritis** the anæsthesia is an early symptom, and in mild cases may be the only one apparent. If the habit is continued the paralysis appears, and is characteristically distributed to the feet and hands, producing the phenomena of *foot-drop* and *wrist-drop*. The majority of cases occur in women. I have often queried in my own mind whether some of the mysterious cases of anæsthesia, with pains, resisting all treatment, would not, if all the facts were known, be relegated to this class of cases.

Plumbic neuritis exhibits *double wrist-drop*, and a history of previous attacks of lead-colic.

Arsenical neuritis is attended by anæsthesia in the extremities of the limbs; but the paralytic phenomena are the most prominent features, consisting of weakness of the extremities, muscular atrophy, and diminished reflexes. The nerve-trunks may exhibit pain on pressure, and the muscles are not infrequently sore to manipulation, even when gently performed.

Beri-beri is the endemic form of neuritis. It has been divided into three types, known, respectively, as the atrophic or dry, the hydropic or wet, and the acute pernicious types. The *atrophic* type presents the ordinary symptoms of a severe multiple neuritis, as severe pains and muscular weakness succeeded by atrophic paralysis. The *hydropic* type presents the same symptoms as the atrophic, with the addition of œdema and circulatory disturbances. The *pernicious* type is characterized by the symptoms of the foregoing, which progress to a fatal termination with peculiar malignancy.

Post-influenzal neuritis produces a most varied symptomatology. Probably the most frequently observed are those exhibiting some degree of numbness as the only symptom, which disappears after a short time. Then we have another class in which there is severe pain, as seen in certain cases of sciatica. Lastly, we may have more or less widely distributed paralysis as in other cases of neuritic palsy. The prognosis of influenzal neuritis is better than for any other form of the disease of equal severity.

The **anæsthesia of defective circulation** is commonly associated with cardiac disorders. It is usually described by the patients as sensations of numbness. In many of the cases it is the result of high vascular tension, which lessens the blood-supply to the peripheral nerve-endings. Occurring in the left hand and arm, it is one of the characteristic features of paroxysms of *angina pectoris* and other so-called cardiac neuroses.

The **anæsthesia from exposure to cold** is due to the double effect of the cold upon the capillaries interfering with their circulation, and the benumbing effect of refrigeration upon the peripheral nerve-endings.

The influence of **tea** in producing anæsthesia and sensations of numbness has not been sufficiently estimated. One finds the acroparæsthesia, described in the next paragraph, very common among women who are excessive tea-drinkers.

The **acroparæsthesiæ** constitute the most frequently-observed examples of sensory deficiency. It is also known as "*waking numbness.*" While described as a numbness by the patient, the symptom is really one of perverted, not deficient, sensibility in the majority of cases. It occurs with especial frequency among women of middle age, and doubtless has much to do with the neurotic instability of that time of life. Dana, who has given the subject considerable study, affirms that the disease is most common among those who are obliged to work a great deal with the hands, as in the case of seamstresses and washerwomen. With this view I cannot coincide, for it is a common source of annoyance of neurotic women of the upper classes who have never done any work worthy of the name. There can be no question of the fact that auto-intoxication is, by reason of high living and deficient exercise, gout, lithæmia and rheumatism, an important constitutional cause. Some of the cases are really due to a light grade of neuritis, especially those having an alcoholic origin. The abnormal sensations are, as a rule, widely distributed. Nevertheless, in many instances they are readily determined to be limited to the areas of distribution of some nerve-branch. Thus patients will speak of them as involving the little and outer side of the third finger. Sometimes the numbness is limited to the finger-tips; in others it spreads over the entire arm. Patients declare that there is loss of motor power, but this is extremely doubtful. The mental perturbation accompanying such of the paroxysms as occur at night gives rise to many morbid fancies, under the influence of which, the patient's ability to give reliable testimony is exceedingly doubtful. Very often there will be accompanying symptoms of dyspepsia, rheumatism, anæmia and constipation, which no doubt in some cases tend to aggravate the neurotic disturbances. While the arms are the most frequently attacked, the head and the lower extremities do not escape; indeed, in some instances the sensory disturbance may be limited to the latter location. The diagnosis of acroparæsthesia should not be difficult, and yet it is occasionally confounded with neurasthenia, multiple neuritis, the occupation neuroses, and organic disease of the brain and spinal cord. Attention to the principles involved in determining the presence of these several conditions will surely guard against error.

Leprosy, especially in the variety designated *anæsthetic*, may be associated with diminished sensibility. The sensory symptoms may present themselves in great variety. In some instances there is a feeling as if the hands and feet are "asleep," or there may be actual anæsthesia. Not infrequently the sense of touch is preserved, that of temperature being lost. This fact has led some authorities to suggest that syringomyelia is the pathological lesion of leprosy. Sometimes the onset of the anæsthesia is marked by delayed sensation, the patient not giving evidence of a sensory perception until twenty or thirty seconds after the stimulus was made. Cases have been observed in which the anæsthesia existed in patches, and was not discovered until a special investigation of the sensory functions had been made. These patches may or may not coincide with eruptive lesions. Sometimes they are surrounded with zones of anæsthesia. The anæsthetic patches are not necessarily permanent, for cases have been observed in which not only has sensation returned, but the parts have become hyperæsthetic.

Diseases of the skin are capable of producing anæsthesia by reason of the destruction (temporary or permanent) of the delicate structures necessary for the perception and conduction of sensation.

Visceral Anæsthesiæ are nearly always of functional or hysterical origin ; exceptionally, they may be organic. Those most frequently observed in practice involve the throat, rectum, bladder and vagina.

Anæsthesia of the throat may occur in

a. Hysteria.
b. Post-diphtheritic paralysis.
c. Bulbar paralysis.
d. As part of a general hemianæsthesia.

Rectal anæsthesia is manifested by a failure on the part of the patient to recognize the presence of fæcal accumulations in the rectum. Constipation from absence of the natural stimulus to stool is the result. It may occur in

a. Hysteria.
b. Locomotor ataxia and other organic affections of the spinal cord.
c. Widespread cerebral degeneration.

Bladder and vaginal anæsthesiæ are found in

a. Hysteria.

b. Affections of the spinal cord, especially myelitis and locomotor ataxia.

Vaginal anæsthesia is usually manifested by complete loss of pleasurable sensation during intercourse. **Bladder anæsthesia,** by the absence of the usual sensations present when the bladder is distended with urine. Bladder anæsthesia is a frequent cause of retention of urine in hysterical subjects.

Hyperæsthesia.—Strictly interpreted, hyperæsthesia should mean an abnormal acuteness of the sense of touch. This term, however, is used to indicate abnormal sensitiveness to pain or touch. That is to say, sensations which in health are transmitted as such, when hyperæsthesia is present give rise to painful impressions. Aside from nervous conditions, it is found as one of the phenomena of inflammation, the affected part resenting slight manipulations, in many cases, even of the gentlest character.

Hyperæsthesia may be general or local. When local it may involve some one particular organ or structure, or some general portion of the body.

General hyperæsthesia of the surface of the body occurs in

a. Hysteria.
b. Hydrophobia.
c. Cerebro-spinal fever.

Hemi-hyperæsthesia or hyperæsthesia of one lateral half of the body is rarely observed. It may occur, however, as a manifestation of

a. Hysteria.
b. Disease of the pons varolii.

Of the **local hyperæsthesiæ,** the most interesting and of the greatest practical importance are those involving many of the viscera,—**sensory visceral neuroses.** The sensory neuroses of the female sexual apparatus in particular lead to many diagnostic errors, as they constitute the principal foundation for the alleged presence of "inward trouble" among women, and, in consequence, lead to the inauguration of most absurd therapeutic measures.

These **sexual neuroses** are referred to the ovaries, the uterus and vagina. Patients complain of spontaneous pain or discomfort or sensitiveness to deep or slight pressure in these regions. Physical examination fails to discover local objective conditions sufficiently marked to account for the severe subjective symptoms. Indeed, I might say that these same subjective phenomena are generally absent in patients who are the victims of a substantive disease, and that the advocates of the genital origin of many nervous complaints in women naïvely contend that it is the very slight or doubtful local lesions which produce reflex disturbance. The presence of the well-known stigmata of hysteria should make the nature of the case clear. But, unfortunately, gynæcological tradition finds a ready explanation for this state of affairs, and demonstrates to the mentally astigmatic gynæcologist that the general nervous manifestations are reflex disorders originating in the uterus and ovaries. The damage done by such effete ideas is incalculable, as many a young life has been ruined by the sentence to the "chair" and pessary. Hysterical patients are proverbially susceptible to suggestions,—for good, as well as for evil. For evil, when informed on presumably high authority that they are the victims of "womb-trouble;" for good, by treatment of any kind whatsoever when practised by a physician in whom they possess confidence. Hence it is that the therapeutic measures directed to the local trouble relieve for awhile, but the condition of the general nervous system remains the same until measures specifically designed for its strengthening are instituted.

The **surface hyperæsthesias of hysteria** are observed especially along the back, being manifested as **sensitiveness of the spine** (so-called **spinal irritation**), over the chest (leading to the absurd diagnosis of cardiac and lung disease), in the breasts (hysterical mastodynia), over the abdomen, and in the hypochondriac region.

Organic hyperæsthesias are observed in lesions involving the meninges of the brain and cord, and in the early stages of peripheral neuritis. Thus we find a band of hyperæsthesia at the upper level of the lesion in spinal meningitis, acute transverse myelitis, caries of the vertebræ, and malignant disease of the cord or vertebræ. This hyperæsthetic area is usually referred to by the patient as "a sensation as of a band around the body,"—*girdle* or *cincture sensation*. It may be demonstrated objectively by passing a hot sponge down the spine, or by the application of the negative electrode of the galvanic battery. This hyperæsthesia from spinal-cord disease is not, however, limited to the trunk, as it may be manifested in the extremities when the posterior spinal nerve-roots tributary to the same are irritated or inflamed.

Hyperæsthesia of muscles and peripheral nerves occurs in peripheral neuritis, whether simple or multiple. Muscular hyperæsthesia is especially liable to occur in the multiple neuritis produced by alcoholic excesses.

Local hyperæsthesia accompanies many cases of neuralgia (*points douloureux*). These sensitive points are usually over the place at which the affected nerve approach the nearest to the cutaneous surface. More will be said on this subject when speaking of the subject of neuralgia.

Paræsthesiæ.—The term "paræsthesiæ" is used to designate perverted or altered sensation. The symptoms included under this head are often referred to by patients as pain, notwithstanding the associated statements belie the accuracy of that designation. The distinction between paræsthesiæ and pain is a most important one from both diagnostic and therapeutic standpoints. To force this distinction upon patients is not an easy matter, unless one puts a stop to their narration of symptoms and tersely asks, " Is it a distress or a pain from which you suffer?" There are very few who will not grasp the distinction at once, when stated in this way.

Paræsthesia may be symptomatic of either organic or functional disease, the latter being the case in the majority of instances. The morbid sensations generally included under this head are the following: Numbness or dead-feeling, itching, formication, tingling, sense of fullness, coldness or chilliness, flashes of heat, faintness, oppression, anxiety, præcordial constriction, throbbing, tightness and weakness.

As an expression of so-called functional disturbance, paræsthesiæ are observed in hysteria, neurasthenia, hypochondriasis, melancholia and other forms of insanity, moderate anæmia, gastro-intestinal disturbance, hepatic inadequacy (lithæmia), and general malnutrition.

While the relation between paræsthesia and mental disturbances is well known to neurologists and alienists, it is not sufficiently appreciated by practitioners at large. Concerning this subject, I doubt if it is possible to speak too strongly. Chilly sensations are diagnosed too often as malaria; numbness as the beginning of organic palsies; visceral sensations as local disease of the particular organ to which the symptom is referred. But the damage done to the patient does not stop with the mistaken diagnosis. If it did, the physician's reputation for skill alone would suffer. But the unfortunate possessor of the symptom is too often the victim of an unstable nervous organization, and the belief in the existence of organic disease so preys on the mind as to render it more unstable, and leads to far more serious disease. Aside from this is the negative damage in failing to institute the proper treatment, which in the beginning means the simple change of scene, or freedom from business and household cares and worries.

As to the diagnostic significance of special paræsthesiæ, no one has written more to the point than Butler, from whom the following is quoted, with a few comments of my own:

"**Bearing Down.**—The sensation of bearing down is felt chiefly in the pelvis. The causes of this sensation are for the most part diseases or conditions affecting the uterus, especially membranous dysmenorrhœa. Hæmorrhoids, undue fullness of the bladder, and sometimes prolonged standing, may also be responsible." The causes producing this symptom are especially active in the presence of anæmia and malnutrition. Indeed, these may be the sole cause, as evidenced by their disappearance on the improvement in the general health. Valuable as this symptom is as a subjective evidence of uterine disturbance, other than local causes must be recognized.

" 2. **Coldness or Chilliness.**—Subjective sensations of coldness, existing with a normal or subnormal temperature, may be due to hysteria, lateral sclerosis, myxœdema, neurasthenia, or syringomyelia; and chilliness may be present as one of the prodromal symptoms of an attack of migraine.

"**Cold sensations (psychro-paræsthesias)** may implicate a whole extremity or all four extremities without being limited to certain areas or the distribution of a nerve (Dana). Not infrequently these are associated with other paræsthesias (prickling and numbness), with pain and with local disturbance of the circulation. This form of cold sensation is usually indicative of a mild neuritis due to the abuse of alcohol, or to a rheumatic diathesis, exposure and toxæmias. It may also occur in locomotor ataxia and in the early stage of syringomyelia.

"On the other hand, the cold sensation may be limited to some special area, usually upon the buttock or thigh, sometimes the calf; corresponding pretty accurately to the distribution of a nerve. The feeling is as though something tangible and cold were in contact with the part. The sensation may be so distressing that it is very properly termed a cold pain (psychro-algia). These circumscribed areas of coldness are found, as a rule, in patients over 40, more commonly in men. The causes may be found in pressure upon the nerves due to occupation, rheumatism, or exposure, and in many cases a neuropathic diathesis is an underlying agency. If obstinate, syringomylia, or rarely locomotor ataxia, may be responsible for this symptom." Too often these sensations are described by patients as nervous chills,—a diagnosis that is too readily accepted by the practitioner. The harm comes when the sensations are symptomatic of actual organic processes, especially of the true chills in the early stages of septic processes. In addition to the causes of cold sensations enumerated by Butler is the circulatory and nervous disturbance incident to *change of life*.

"3. **Faintness.**—A sensation of weakness with a tendency to syncope, persistent, recurrent or acute, may be present as an accompaniment of the various forms of anæmia, angina pectoris, fatty heart, thrombosis of the pulmonary artery, pneumothorax, thoracic aneurysm, ascites (especially during tapping), and tympanites. It may be present as the result of emotion, fatigue, excessive heat, painful affections, depressing poisons, and as evidence of shock after injuries. As an idiosyncrasy, it attends the act of defecation in certain persons, and is a frequent event in Meniere's disease." In addition to the above, I would direct attention to fainting as a clinical manifestation in other cardiac affections than angina pectoris and fatty degeneration. It is especially important as a feature of cardiac neuroses (palpitation, functional tachycardia, bradycardia). Flatulent distention of stomach and intestines, especially in neurotic subjects, accounts for a large class of cases which have tried the therapeutic resources of many physicians. The foul air of crowded assemblies is responsible for attacks in healthy though not overly-strong persons. Some women experience faintness from the fœtal movements during pregnancy.

"4. **Formication, Itching.**—Formication is a sensation as of ants or other insects crawling over the skin. It is a variety of itching or pruritus, and, in varying degrees and in different portions of the body, may result from many diseases of the skin, including the exanthemata. Affecting the external genitals, it may be due to diabetes, leucorrhœa, or neuroses; the anus, to seat-worms or hæmorrhoids. It is an occasional premonitory symptom of apoplexy, and may be present as a consequence of tumor of the brain, in the part supplied from the seat of lesion. More or less general pruritus frequently occurs in hysteria and neurasthenia, and also in locomotor ataxia, chronic spinal meningitis, chronic myelitis, disseminated sclerosis, chronic lead-poisoning, and tetany. It is general and frequent in jaundice; less often it is found in gout and cases of granular kidney. It is an occasional result of the administration of morphia (especially of the nose), copaiba and ergot.

"General itching or pruritus, if persistent, should lead to an examination for the existence of gout, lead-poisoning, chronic interstitial nephritis, or diabetes mellitus." Formication and itching are not uncommon results of the toxæmia arising from constipation. Formication especially is a manifestation of climacteric neurasthenia; indeed, it is observed as frequently as are the hot flashes and cold chills of that period of life. Some persons experience intolerable itching and formication from woolen underclothing. I have once seen these symptoms appear as part of the phenomena of chronic arsenical poisoning.

"5. **Fullness.**—A sensation of fullness in the chest or epigastrium is caused by chronic gastritis, nervous dyspepsia, or dilatation of the stomach, as well as by cardiac hypertrophy, pulmonary emphysema, chronic peritonitis, tympanitis, or malarial fevers.

"6. **Girdle Sensation.**—A subjective sensation, as of a constricting band around the trunk, is found in locomotor ataxia, chronic myelitis, injury or tumor of the spinal cord, and inflammation or tumor of the spinal meninges. After prolonged vomiting or repeated paroxysms of violent cough a similar sensation may be felt, corresponding to the insertion of the diaphragm, and due to a strain of that muscle." We think that too much dependence must not be paid to the cincture sensation as a symptom of locomotor ataxia. It is distinctly a symptom of meningeal irritation, and cannot occur in uncomplicated tabes.

"7. **Heat.**—Subjective sensations of heat may be felt in the epigastrium and beneath the sternum in pyrosis and irritant poisoning, and to a less degree in acute bronchitis; in the back in spinal irritation or neurasthenia. Flashes of heat, with or without flushing of the face, and sudden perspiration, are common attendants of the menopause, as well as disease of the pelvic viscera and conditions of debility and nerve-tire. Subjective sensations of heat may be felt in the back and epigastrium in paralysis agitans.

" 8. **Numbness, Tingling, Burning.**—These are of rather common occurrence, and may possess diagnostic value. Particularly in the feet, they are in some cases the first symptoms of locomotor ataxia, myelitis, or chronic spinal meningitis, and are very frequent in neurasthenia and hysteria, and (in the fingers and toes) are not infrequent in arthritis deformans. Numbness (fingers, occasionally scalp and feet) on waking in the morning, which at first passes away, but later remains during the day, is *acro-paræsthesia*, or *waking numbness.* Localized numbness is sometimes caused by a tumor of that portion of the brain which supplies the area of abnormal sensation. In tetany there is numbness of the fingers and toes, and tingling or burning is an occasional premonitory symptom of neuralgia or herpes zoster. Certain poisons may also cause numbness.

" 9. **Oppression or Weight.**—When felt in the chest, is most frequently found in hæmoptysis, hysteria, spasmodic asthma, angina pectoris, nervous dyspepsia and chronic gastritis ; less commonly in cardiac or pericardial disease, Basedow's disease or exophthalmic goitre, and tumor of the mediastinum. When felt in the epigastrium, is usually caused by neurasthenia, chronic gastritis, nervous dyspepsia, or, rarely, as a premonition of gastric hæmorrhage. A feeling of pressure or weight in the head is common in neurasthenia or hypochondriasis, and is occasionally premonitory of apoplexy, or is a part of the epileptic aura. A similar sensation in the *abdomen* or *pelvis* attends the presence of abdominal or pelvic tumors."

" 11. **Throbbing.**—This, as a subjective sensation, felt over the entire body or in the head and extremities, is not infrequent in hysteria, neurasthenia, vaso-motor ataxia, the anæmias, and aortic regurgitation. Locally it is felt in phlegmonous inflammations and over the seat of aneurysms ; in the neck and head in cases of exophthalmic goitre ; in the head in migrainous headaches ; in the præcordial area in cardiac hypertrophy or palpitation ; and the throbbing aorta is very common in gastritis and neurasthenia."

" 12. **Tightness.**—This, when substernal, is usually associated with the dry stage of acute tracheitis and bronchitis of the larger tubes. The term is also applied to the character of the cough in the same stage.

" 13. **Weakness or Debility.**—A feeling of muscular weakness, debility or lassitude attends the prodromal stage of fevers, and is a sequela, more or less prolonged, of all febrile and exhausting diseases. It may also be a concomitant of the dyspepsias ; the overuse of tea, alcohol and tobacco ; and rheumatism, gout and lithæmia.

"This symptom, when persistent, has a distinct clinical value in suggesting the presence of neurasthenia (most frequent), pulmonary tuberculosis (even without physical signs), diabetes, anæmia, influenza (obstinate), latent pleurisy, and exophthalmic goitre."

Pain.

This consideration of pain as a symptom is to be limited to its study mainly as it occurs in connection with nervous diseases and nervous conditions. Pain accompanying inflammatory and other states manifested by local objective phenomena, as heat and swelling, finds elucidation elsewhere. In practice we find personal susceptibility to pain to vary greatly. Some persons bear with it with great fortitude,—suffering greatly, and yet making no complaint. Others seem to have the pain sense more or less blunted, and really suffer but little from conditions which in others would give rise to the greatest agony. Then we find people at the other extreme. A small scratch or the slightest aching calls forth the loudest lamentations. Some magnify their pains by catering to them, as, for example, is seen so frequently in the hysterical, whose quiet, placid facial expression contradicts plainly the intensity of their sufferings as so graphically depicted by them in spoken words.

The pains with which we have to do at this time are those arising from the following causes:

1. Neuralgia.
2. Neuritis.
3. Disease of the spinal cord.
4. Reflex pains.
5. Pains from various toxæmiæ.
6. Rheumatic and gouty pains.
7. Hysterical pains.
8. Anæmia.

(1) **The pains of neuralgia**, as this condition is usually understood, are characterized by their paroxysmal character in the majority of cases. Still they may be continuous, in which case they are generally dull, and often interrupted by acute exacerbations. They are often associated with the painful points first described by Valleix, and which are situated, speaking in a general way, over points where the nerve emerges from its bony canal, enters a muscle, where its trunk approaches close to the surface of the body, and at the termination of the nerve-branches near the surface of the body.

(2) **The pains of neuritis** are limited to the course and distribution of the affected nerve or nerves. Tenderness is experienced along the nerve-trunk. The pain may be dull in character, or sharp, shooting and boring. It is greatly aggravated by causes that put the affected nerve on a stretch, and is often intensified at night, and by changes in weather to cold or damp.

From a clinico-pathological standpoint, it is a difficult matter to determine just what cases shall be relegated to the classs of neuralgias. The definition of neuralgia provides for a state of affairs that must be rarely observed in practice, if one is careful in making thorough and systematic examinations. This definition is a nerve pain occurring independently of any changes in the nerve structure, and occurring independently of organic lesions in any other portion of the body. We find by experience that nearly all cases of so-called neuralgia are examples of neuritis or nerve degenerations, or else are reflex or toxic in origin. As a matter of convenience, we may continue to use the term, but we should do so with considerable circumspection.

The study of the nerve pains in different portions of the body may best be undertaken if, for this purpose, we make neuralgia and neuritis synonymous terms.

(3) **Pain as accompanying disease of the spinal cord** is very much misunderstood by practitioners generally. It is altogether too common a custom to ascribe many severe spinal pains to this cause, when in reality such cases are of rare occurrence. It must be remembered that backache may occur from a great variety of affections, as disease of the pelvic viscera, gastric and hepatic disorders, neurasthenia, hysteria, disease of the bone and spinal membranes, and chronic inflammation of the fibrous tissues of the spine. Those pains which originate in functional disturbances, as the sensitive spines of neurasthenia, hysteria, etc. (and generally spoken of as spinal irritation), are readily recognized by their tendency to shift from one part of the vertebral column to another, while those arising from organic causes are very likely to be fixed. Organic spinal pains are especially liable to arise from meningitis and disease of the vertebra. In the former the sensitiveness is experienced over the bones themselves, and in the latter, between the vertebra. They are apt to be especially severe when arising from malignant disease of the bones or meninges. Chronic organic diseases of the cord do not often give rise to severe local back pains.

Peripheral or eccentric pains very frequently occur as spinal-cord disease. They may arise (1) from irritation of the sensory nerve-roots as they pass through the posterior columns of the cord, through the membranes, or the intervertebral foramina, and (2) from irritation of the sensory-conducting tracts in the cord. The eccentric pains are almost always bilateral and symmetrical. They are often of a severe darting character, and their distribution generally indicates the height of the lesion in the cord. The pain from disease of the sensory-conducting tracts is generally dull in character, and is very liable to be confounded with the pains of rheumatism, especially as it is very often aggravated by changes to damp or cold weather. A very characteristic pain accompanying spinal-cord disease, one, too, that is regarded by many neurologists as positive proof of the existence of organic changes in the cord or its membranes, is the so-called "cincture feeling," or "girdle pain." It is a sensation as of a band around the waist or trunk, and is caused by irritation of the nerve-roots at the lowest portion of the healthy part of the spinal cord. It is found in the different forms of myelitis and meningitis, and in some cases of spinal sclerosis. It sometimes occurs in the lower extremities, then being manifested by a sense of constriction, and is bilateral and symmetrical.

4. **Reflex Pains.**—The term "reflex," as applied to pain, is intended to convey the idea that the pathological condition giving rise to the symptom is situated at some other portion of the body than that in which the pain is experienced. Many notable examples of reflex pain may be noted. Thus we have:

Pain at the end of the penis in stone in the bladder.

Pain in the testicles, extending even into the thighs, from renal calculus.

Pain about the inner side of the knee from disease of the hip-joint.

Pain on top of the head from uterine disease, especially endometritis and bladder inflammation.

Pain over the clavicles from diaphragmatic disease.

Pain over the left border of the sternum from gastric disorder, and often described as pseudo-angina.

Pain in the mammary glands from uterine disease.

Pain in the left hypochondrium from constipation and indigestion.

Pain at the angle of the right scapula from disease of the liver.

Pain over the fourth and fifth dorsal vertebræ from disease of the stomach, especially, however, from ulcer.

Pain over the seventh to ninth dorsal vertebræ from disease of the colon.

Pain over the last dorsal and all the lumbar vertebræ from uterine disease.

Pain over inner and upper portions of thighs from disease of the ovaries.

These reflex or transferred pains, as they are also called, are too frequently diagnosed as neuralgic, without any attempt to determine their real source having been made.

5. **Pains from Various Toxæmiæ.**—The dictum that constitutional causes produce general or bilateral symptoms has already been quoted. In accordance with it we would expect toxæmic pains to be generalized, or else bilaterally but symmetrically distributed. The most notable examples of the toxic pains are found in acute diseases, especially in those of the infectious type. We find generalized aching a common attendant upon the early stages of typhoid fever, small-pox, etc. The most commonly observed example, however, is found in follicular tonsillitis, which is almost

invariably attended at its onset by severe aching pain, involving almost every portion of the body, and aggravated by any motion. It is not unusual for the patient to say that he feels as if he had been pounded or put through a rolling-mill. The chronic toxæmia do not seem to be capable of producing actual pain without the intervention of actual lesions, such as neuritis and vascular changes.

Hysterical Pains.—There is not, in the whole category of disease, a single affection which does not have its hysterical counterfeit; and this remark applies with equal force to "pain." To describe in words the characteristic features of an hysterical pain is impossible, if one wishes to give his reader an adequate conception of the condition. To my mind, the best plan when attempting a diagnosis is to have in mind the actual organic affections which are simulated by the case in hand, and then note in what particulars the symptoms presented fall short of establishing the unequivocal presence of the suspected conditions. If they fail to do so, it is reasonable to suspect a hysterical origin. Thus we have a diagnosis by exclusion. But this is not sufficient; for practically all cases of hysteria, if observed carefully for a sufficient length of time, will exhibit the well-known stigmata of that disease, if disease it may be called; and in the case of pain, the patient's facial expression and her general constitutional condition are not consistent with the great intensity of the pain as described by her. The hysterical hypothesis is still further favored by the presence of an emotional cause, and the age and previous history of the patient. It does not always follow, in hysterical conditions, that what the patient calls a pain is actually such. It is sometimes but a morbid sensation,—a "misery," to borrow a word in common use among the colored race in describing their sufferings. In neurasthenia, the allied condition, these morbid sensations are the rule; true pain, the exception.

I cannot illustrate my remarks better than by referring to a patient recently seen in consultation. The patient, a young unmarried woman, complained of severe pain in one thumb. Her suffering becoming agonizing, periosteal disease was suspected, and a deep incision was made without bringing the expected relief. The wound healed; the pain continued, but there were no local objective changes to account for the pain. She then displayed marked mental disturbance; her wanderings were decidedly hysterical in type. When I saw her, she had been kept under the influence of large doses of morphia hypodermatically (two to three grains daily), and still the pain continued. The diagnosis at this time was neuritis. It was inconceivable to me that pain limited strictly to the thumb, involving equally its entire circumference, segmental in character (it stopped short at the base of the member), without local tenderness, could be due to a neuritis. There was no history of any cause capable of producing a neuritis. Firm pressure and manipulation of the thumb produced no discomfort. The mental condition and the absence of positive signs of local disease established a diagnosis of hysteria, in which the attending physician concurred, in a letter received by me a week subsequent to the consultation.

6. **Rheumatic and Gouty Pains.**—Rheumatic and gouty pains are, for the most part, fixed pains. They are especially liable to be confounded with the pains of traumatism. It must be remembered that very often a sprain of a joint or a traumatism may serve to fix a rheumatic pain to a definite locality, and so occasion errors in diagnosis. While rheumatic pains are nearly always aggravated by changes in weather, the pains of neuralgia, neuritis and organic disease of the cord are often subject to the same modality. One cannot, then, be too careful in all doubtful cases to study the case as a whole, and not to jump too quickly to the conclusion that rheumatism is the trouble.

Sudden removal of a long-continued pressure will often excite much agonizing pain. The best example of this is found in the severe pains in the legs following rapid delivery.

7. **Anæmic pains** usually answer to the description of the so-called neuralgias. In some cases they are truly hysterical, having been the result of anæmia acting in a subject of hysterical temperament. The diagnosis is based upon the recognition of the anæmia.

As to the diagnostic value of the character of pain, Leftwitch offers the following suggestions, which, however, must not be accepted as invariably having the significance stated by him:

Sharp pains:

Angina pectoris; appendicitis; dissecting aneurysm; ectopic gestation; gout; pleurisy; pneumothorax at the onset; spinal hæmorrhage; acute inflammations of serous membranes in general.

Dull pains:

Chronic inflammation of serous membranes; inflammation of mucous membranes and visceral parenchyma.

Paroxysmal pains:

Angina pectoris; aneurysm; appendicitis; colic; cancer of the pancreas; calculus of the pancreas; cerebral tumors; clot in ureter; distended bladder; dysentery; dysmenorrhœa; floating kidney; hydatids of the kidney; hepatic colic; lead colic; lumbrici; locomotor ataxia; mercurialism; neuralgia; parturition; renal calculus; sporadic cholera; scybala or intestinal concretions; strangulated hernia; tubercular synovitis; tubal colic; vesical calculus; urethral calculus; uterine fibroids; uterine polypus.

Radiating pains:

Acute aortitis; angina pectoris; aneurysm of the ascending aorta; abdominal aneurysm; aneurysm of the innominate artery; atony of the stomach; caries of the spine; compression of the spinal cord; gastritis; hepatic colic; hip disease; neuralgia; oöphoritis; pancreatic calculus; pelvic abscess; pachymeningitis; renal calculus; cancer of the rectum; spinal tumor; spinal hæmorrhage; spinal meningitis; uterine fibroids; vesical calculus.

Shifting pains:

Flatulence; hysteria; locomotor ataxia; spinal-cord tumor; tape-worm; trichinosis.

Gnawing pains:

Abdominal aneurysm; caries of the spine; cancer of the stomach; descending thoracic aneurysm; gout; lithæmia; periostitis; spinal meningitis.

Pain increased by movement:

Acute inflammatory diseases; abscess; caries of the spine; fractures; gout; glandular fever; lumbago; neuritis; oöphoritis; external and internal pachymeningitis; perinephritis; acute pleurisy; pneumonia; pleurodynia; acute rheumatism; muscular rheumatism; sciatic neuritis; spinal meningitis; spinal-cord tumor; salpingitis; Weil's disease.

Pain increased at night:

Locomotor ataxia; neuritis; osteitis; periostitis; renal calculus; rheumatism; syphilis.

Before proceeding with a study of pain as related to special portions of the body, it is convenient to mention two conditions not frequently observed, in which the pains may be assigned to any part, or, indeed, may be general. These affections are akinesia algera and adiposis dolorosa.

Akinesia Algera.—This is a condition characterized by the inability to move a part because of the presence of pains which are not to be explained on any other hypothesis than their psychopathic origin. Möbius, who first described it, has endeavored to still further extend the scope of the mental state calling it forth by substituting the term "apraxia algera," this including all conditions characterized by "abeyance of the function of any or all organs, because of pain attending their functional activity." So far as akinesia algera is concerned, it is a rare disease. If we accept the term "apraxia algera" as including psychopathic pain attendant upon visceral function, it is very common. The disease attacks persons of neuropathic constitution, especially those possessed of paranoiac inheritance. It is of gradual onset. Usually the patient gives a history of psychoses extending over a term of years. Pains come on, and are complained of as being intense. At the same time, the most careful examination fails to account for their presence. Eventually all the muscles of the body are involved. Objective disturbances, as anæsthesiæ, muscular atrophy, altered electrical reactions, etc., are absent. The pains are believed to be pure hallucinations. They are greatly aggravated by the slightest motion, which truly causes the most intense suffering. The patient, therefore, lies in bed motionless and helpless. No alterations in the reflexes have been observed. Dana, in his description of the disease, places it among the hysterical affections. The majority of observers regard it as a form of insanity.

Adiposis Dolorosa.—This affection was described by Dercum in 1892. It is characterized by the irregular appearance of fat in nodular masses in various portions of the body. It is the rule for the enlargement to appear in some one situation, or perhaps in symmetrical parts—usually in the arms or legs. At some time or another in the course of all the cases pain is a prominent feature, and is usually of a shooting, burning, or stabbing character. Other nervous symptoms manifested include anæsthesia and weakness. Some of the cases have been attended by hæmatemesis, epistaxis, metrorrhagia, and other hæmorrhages. Dercum believes the affection to be a connective-tissue dystrophy, "fatty metamorphosis of various stages of completeness occurring in separate regions, or at best unevenly distributed, and associated with symptoms suggestive of an irregular and fugitive irritation of the nerve-trunks—possibly a neuritis."

Pains of the Head. Headache.

The frequency with which the physician is consulted for the relief of headache and the fact that head-pains ofttimes constitute the first symptom of serious diseases make it a matter of such importance that I shall treat of this subject rather elaborately. The early stages of many of the acute ailments are sometimes preceded for some days by headache; and although these as well as the cephalalgias arising from certain toxæmias are in no sense nervous ailments, still their neurological relations are such as to make their consideration in this place perfectly proper.

In no class of ailments is the question of etiology of more importance than in the one now under study; in fact, I might say that, given the cause of the headache in a given case, its prompt cure becomes a comparatively easy matter. I shall endeavor, therefore, to follow a classification based on etiology.

In the clinical study of any case of headache, there are certain points that must be carefully inquired into. Is the pain constant or paroxysmal? If the former, does it vary much in its intensity? If the latter, what is its frequency, and what is the usual duration of the attacks? Is it a pain, or a mere uncomfortable sensation? Do the paroxysms have a regular cycle, never to be departed from? What is the character of the pain, whether throbbing, sharp, dull, etc.? What is its location? What are its associated symptoms? How is it affected by certain actions on the part of the patient, as use of the eyes, change in posture, sneezing and coughing, pressure over the seat of pain, and how by certain extraneous agencies, as weather, light, noise, heat, and cold? As pertaining to associated symptoms, it is especially important to investigate the condition of vision, and the presence or absence of optic neuritis and errors of refraction, to inquire concerning the digestive functions, the presence or absence of nausea and vomiting, and the association of certain nervous disturbances, as numbness and tingling, exhaustion, etc.

The character of the pain offers us but little help in determining its origin; of more importance are the associated symptoms and the modalities that ameliorate or aggravate the ailment.

In presenting a classification of headaches based on their etiology one must necessarily tread on debatable ground, for certain headaches are variously classified by equally eminent authorities. Thus the headaches associated with gastric disorder are regarded by some as toxæmic, and by others as purely reflex; those of contracted kidney as toxæmic (uræmic) and circulatory. The following classification is not offered as one absolutely correct, but merely as an aid to clinical study:

1. Headaches symptomatic of organic disease of the brain and its membranes, as:
    - a. Brain tumor.
    - b. Brain abscess.
    - c. Meningitis.
    - d. Hydrocephalus.
    - e. Intracranial hæmorrhage.
    - f. Dural disease.
2. Circulatory. Including
    - a. Congestive.
    - b. Anæmic.

Under this head may also be considered certain of the headaches accompanying contracted kidney, when such are not uræmic, but are purely the result of the increased arterial tension, and certain cases arising from traumatism.

3. Headaches originating in certain toxæmia, as:
    - a. Uræmia.
    - b. Lithæmia.
    - c. Rheumatism.
    - d. Septicæmia.
    - e. Diabetes.
    - f. Malaria.

  - g. Syphilis.
  - h. Gastric disorders with retention and decomposition of food, etc., in the gastro-intestinal tract. Under this head may be included the cephalalgia from constipation.
  - i. Poisons introduced from without, as alcohol, lead, tobacco, and noxious gases.
- 4. Headaches reflex from disease in adjacent or distant organs, as :
  - a. Eyes.
  - b. Nose and throat.
  - c. Ear.
  - d. Intestinal tract.
  - e. Sexual organs.
- 5. Neuropathic, as :
  - a. Epileptic.
  - b. Hysterical.
  - c. Neurasthenic.
  - d. Hemicrania or migraine.
  - e. Certain essential headaches, the causes of which have been hitherto undiscoverable.

It is important that the physician do not confound headache with other painful affections of the head, such as are caused by disease of the cranial bones and their investing membrane, carious teeth, rheumatism of the scalp, and last but by no means least, glaucoma. The mere act of calling attention to osseous and periosteal disease is all-sufficient to prevent diagnostic error where these are concerned. It may be borne in mind that in the majority of cases, but by no means in all, the augmentation of the pain on pressure affords evidence that the disease giving rise to it is extracranial.

Decayed teeth have been known to produce severe headache, mental disturbance, and defective vision. Concerning the importance of the teeth in the production of pains other than toothache I have already spoken when treating of painful affections of the face.

But of all affections, **glaucoma** is one that must be differentiated from headache. A mistake is rendered easy by the comparative rarity of this affection in the experience of the general practitioner, and by its occurrence in constitutions supposed to favor the origin of headaches. The pains may be so located as not to suggest the eyes as their cause. The visual disturbances may be attributed to the same vaso-motor changes that occur in ophthalmic migraine. In glaucoma there is a concentric diminution in the field of vision which is more or less permanent, while in migraine there is a limitation of vision to one-half of the visual field,—a hemianopsia ; or the pain is associated with general obscuration of vision or certain visual hallucinations. In glaucoma there is an increased intra-ocular tension, and ear and nasal cavities, and various infectious diseases. The possibilities of the latter in the production of meningitis do not seem to have been fully appreciated.

The diseases capable of thus acting include pneumonia, variola, typhoid fever, rheumatic fever, whooping-cough, measles, scarlet fever and tuberculosis.

From a symptomatic standpoint it is not necessary to describe the clinical course of tubercular meningitis, for it presents practically the same phenomena as does meningitis in general. The diagnosis must rest upon the previous history of the patient, and the course pursued by the illness.

Until recently, the diagnosis of meningitis could never be assured. The most that we could say was that meningitis was probably present. Since the introduction of lumbar puncture and improved bacteriological methods, we are enabled not only to diagnose meningitis with certainty, but to name the particular microörganism at fault. So far as the lumbar puncture is concerned, that may be performed by any practising physician. The bacteriological examination of the exudate is, however, a matter involving such wide experience in the details of laboratory methods that it is better left to the bacteriologist.

The diagnostic value of lumbar puncture depends upon the fact that the subarachnoid spaces of the brain and spinal cord are in direct communication with each other. It follows, therefore, that an examination of the subarachnoid fluid removed from the lower end of the spinal canal will denote the character of that within the cranium. The operation for its removal is devoid of danger. The needle used will vary in size, according as the patient is a child or an adult. In the former case it should have the calibre of the ordinary hypodermatic instrument, and be at least 5 to 6 cm. in length. In the case of adults it should have a diameter of 1½ mm., and a length of at least 8 cm. The situation for the puncture should be about 5 mm. from the median line, and between the third and fourth lumbar vertebræ in adults, and between the fourth and fifth in children. An aspirator is not required, for as soon as the needle enters the subarachnoid space the effusion begins to flow. Failure to obtain fluid, when present, is generally the result of not having entered the needle to a sufficient depth. The position for performing the operation must be modified according to the age of the patient. In children, the most convenient is with the patient lying on his belly across the lap of an attendant or nurse, thus forcing the back into a convex position. Adults may be placed upon one side with the body bowed well forwards; or, if circumstances permit, they may sit upon a chair with the spinal column flexed upon the pelvis. As soon as the fluid begins to escape it should be caught in a sterilized test-tube, which should be afterwards stoppered with sterilized absorbent cotton.

The general practitioner may examine it for the presence of albumin.

The normal cerebro-spinal fluid contains but small traces of the substance. In the case of meningitis, albumin is present in large amounts,—usually as much as one to two per cent.

The microörganisms found by the bacteriologist will vary according to the cause of the meningitis, and include tubercle bacilli, streptococci, pneumococci, and the diplococci lanceolatus, the latter in the epidemic cerebrospinal variety. The discovery of a large quantity of albumin in the fluid is positive evidence of meningitis; the recognition of specific bacteria names with unerring certainty the cause.

2. **Headaches Dependent upon Alterations in the Vascular Supply to the Brain.**—They may be either anæmic or congestive.

(*a*) **Congestive Headaches.**—In this variety the pain may be either local or general,—usually, however, the latter. In the former event, its point of greatest intensity is in the immediate vicinity of the points at which the large veins enter the longitudinal sinus. The pain is accompanied by subjective sensations of heat, which are proven by means of the surface-thermometer to have an objective origin. The patient's sufferings are aggravated by the recumbent posture, and by repeated acts of coughing. It is well to bear in mind that a cough may apparently of itself give rise to a very persistent headache. In fact, I have seen one case with the headache so much more prominent than the cough that it was the patient's only source of complaint. The history of cough was only elicited after systematic search for the cause of the headache. Usually congestive headaches are associated with marked throbbing of the blood-vessels of the head and neck and suffusion of the face. Da Costa has laid stress on the value of the ophthalmoscope in revealing the circulatory condition. I feel, however, that in this place that instrument is not a reliable guide; for different observers, equally skillful, will present reports at wide variance with each other when they come to describe the size of the retinal vessels. The action of the heart is greatly accelerated. The mental condition is one of irritability.

(*b*) **Anæmic headache** is especially apt to occur after hæmorrhages, as post-partum, in case of hæmorrhoids, and as an accompaniment of constitutional anæmic states. It is frequently met with in chlorosis, and Rake has described a case of pernicious anæmia (*British Medical Journal*, 1885, vol. ii., p. 913) in which it was the patient's only source of complaint.

The pain is usually symmetrical, and situated in the vertex. Sometimes it is located by the patient as immediately back of the orbit, and is described as if the "eyes were being drawn out of their sockets." It is greatly relieved by lying down. In severe cases, efforts to rise may produce syncope. The characteristic anæmic cardiac murmur is present.

3. **Headaches Originating in Certain Toxæmiæ.**

(*a*) **Uræmic Headaches.**—Headache is not an infrequent concomitant of both acute and chronic uræmia. To the former it is a precursor, and lasts but a comparatively short time. It is then the result of any of the forms of kidney disease. As a symptom of chronic uræmia it exists mainly in connection with chronic contraction of the kidney. It may last for many months. The pain may be situated in almost any portion of the head, principally, however, in the frontal and occipital regions. It is, as a rule, quite intense ; and it may be either paroxysmal or continuous. The diagnosis must be made by the urinary examination and the detection of symptoms significant of renal disease. One must not lose sight of the fact that the form of Bright's disease in which chronic headache is the most frequently met with is also the one in which albuminuria is the least prominent. A quantitative analysis of the urine for urea should be instituted in every suspected case.

(*b*) **Lithæmic Headaches.**—The headache of lithæmia apparently has no special diagnostic characteristics. Wood teaches that its usual form is a dull and heavy pain ; while, if we are to believe Haig, it usually assumes the migrainous type. The latter authority almost takes the ground that migraine and uric acid headache are synonymous terms. The paroxysms of pain recur at intervals of a week or ten days, or from that to once a month. It is very rare that individual attacks last longer than from twelve to thirty-six hours. They are especially apt to be brought on by a highly nitrogenous diet, as butcher's meat and cheese, and are greatly relieved, if not entirely cured, by a strict adherence to a vegetarian existence.

The normal relation between the amount of uric acid excreted in the urine and the urea is as 1 : 33. Any cause that serves to increase this ratio may excite the headache. But, as Haig has shown, in order to have a plus excretion of uric acid, there must previously have been a storing up of this substance in the system, as shown by a diminished excretion, as 1 : 40. When the plus excretion comes, it is in exact proportion to the previous retention. Thus the urea-uric acid relation may be as high as 1 : 20, if the preceding retention warrants it. Haig places but little reliance on the actual quantity of uric acid, but only on its relation to the urea.

Accompanying the lithæmic headache is usually a high-tension pulse. Preceding it, is usually a fit of mental depression. Some observers have reported cases in which the opposite mental condition prevailed, the patient being unusually happy.

On the days preceding the headache the urea-uric acid relation is diminished ; on the day of the headache it is greatly increased.

The uric acid headache may begin in childhood, owing to hereditary predisposition or to improper diet.

The gouty headache is but a variety of the lithæmic ; in fact, an advanced stage of the latter. In this case we find ample evidence of gout elsewhere. The headaches are relieved whenever other portions of the body are the seat of gouty invasion. It has been claimed that the headaches of gout are, in reality, of the organic variety, being the result of changes in the brain and its membranes from the patient's dyscrasia.

(*c*) **Rheumatic headaches** are usually of a dull, heavy, aching character. They are especially apt to be associated with soreness of the scalp, and are aggravated during spells of damp or cold weather. In occasional instances they may be the result of rheumatic inflammation of the nerves of the scalp, in which case the pains will partake of the neuralgic character.

Occasionally one meets with a type of rheumatic headache described by Nordstrom. The pains partake of the character of those of migraine. Investigation, however, shows that they depend upon thickening of the insertions of certain muscles, notably the sterno-mastoid and occipito-frontalis. These indurations do not usually give rise to local pain; hence, they are apt to escape observation. They are commonly the result of taking cold. Nordstrom finds an efficient remedy in massage of the indurated spots.

Along with the headaches of septicæmia may be mentioned that ushering in typhoid fever and other low forms of acute disease. As already stated, the fact must simply be borne in mind that such headaches may occur as prodromal symptoms, without other evidence of subjective disease.

(*d*) **Diabetic Headaches.**—Headache does not appear to be an especially annoying symptom during the ordinary course of diabetes. In the later stages of the affection, when acetonæmia supervenes, it is of great severity, and of especial importance in view of the probable onset of diabetic coma. Under these circumstances it is apt to be associated with epigastric pain, gastric disturbance, vomiting, dyspnœa, and delirium.

(*e*) **Malarial Headaches.**—Head-pains due to malaria *per se* are characterized by their periodicity. They are usually situated near one or the other supraorbital foramen, and come on at a fixed hour every day or every other day. They may or may not be accompanied by other evidences of malaria. Sometimes the pain takes the place of one of the typical stages of intermittent fever, chill, fever and sweat.

We sometimes meet with secondary malarial headaches, that is those dependent upon the concomitant gastric disturbances and anæmia of intermittent fever. In such cases, the pain does not present the periodicity referred to.

(*f*) **Syphilitic Headaches.**—A severe form of headache is often met with during the secondary stage of syphilis. The pains are of a boring and splitting character. It generally involves but a portion of the head, and is decidedly worse at night. There is considerable tenderness of the scalp. The headaches of tertiary syphilis are due rather to organic lesions than to any special toxæmia. Syphilitic headache may occur as early as six weeks after infection.

(*g*) **Headache from Gastric Disorder.**—The majority of pains in the head in conjunction with dyspepsia are of a purely reflex character. There still remain a number of cases in which the difficulty arises from decomposition of food in the intestinal canal, and the absorption of the products. The pain in such cases may be very severe, as in a case reported by me in the *Hahnemannian Monthly* for 1886. In this case, so great were the patient's sufferings that organic brain disease was diagnosed. The severe vomiting and the horribly putrid breath suggested a retention of food and decomposition of the same in the digestive tract. Washing out of the stomach with gavage was practised on alternate days. In ten days' time all pain and vomiting had ceased, and the tongue had become clean. After several months' steady treatment the patient was practically cured, aside from an atrophy of the optic nerve, which progressed to complete blindness.

Headache is a very frequent symptom from constipation. This point is one that is altogether too much neglected by members of our school. It is not at all singular that the retention of such foul substances as fæcal masses within the human organism any longer than normal should cause trouble. The only wonder is that carelessness in attention to the bowels does not occasion more difficulty than it does.

(*h*) **Toxic Headaches.**—Of poisons introduced into the system from without, alcohol is the one that most frequently produces headache. It does this not only by its effects on the blood, but also reflexly by first exciting gastric disorder.

Other poisons, as lead, strychnine, quinine, and the narcotics, opium, chloral, etc., may also produce chronic headaches. No special phenomena are attached to the pains from these causes. The diagnosis must be based on a knowledge of the habits of the patient, together with a study of the associated symptoms.

4. Headaches Reflex from Derangement of Adjacent or Distant Organs.

(*a*) **Ocular Headache.**—Ophthalmic difficulty is a very frequent cause of chronic headache. The affections active in this respect are errors in refraction and insufficiencies of the ocular muscles. The former are more frequent causes, simply because they are the more common. Certain forms of muscular insufficiency are almost certain to produce headache. Astigmatism is the most frequent and active refractive error in the production of headache; compound hypermetropic astigmatism is the most important in this respect; simple myopia is the least likely to give rise to such difficulty. Of the muscular insufficiencies, hyperphoria is almost certain to produce headache, not more than 10 per cent. of those subject to it escaping. The degree of refractive error or muscular insufficiency does not seem to bear a definite relation to the severity of the pains. I have seen long-lasting headaches radically cured by the correction of a simple hypermetropic astigmatism of 0.25D; and I have seen cases with a comparatively high degree of refractive error causing only visual disturbance. If there is any rule, it is to find the headaches in persons whose acuity of vision is normal, and in whom there is but a low degree of ametropia. The same remarks apply to muscular insufficiencies. If there is a high grade of weakness the patient soon learns to disregard the weakened eye, and makes no effort to overcome the difficulty.

The pain in cases of errors in refraction is usually found in the frontal region or in the eyeballs. That from muscular insufficiency is located generally in the cervical region or in the occiput. The pain may be either dull or acute; constant or paroxysmal; or, again, it may partake of all the characters of a typical migraine. While in the majority of cases the pain is much aggravated by the use of the eyes, still cases are not infrequent in which the symptoms do not offer the slightest suggestion that the eyes are the seat of the difficulty. Then the only test is a thorough ophthalmic examination.

Difficulties in diagnosis are presented because of the frequency with which these headaches occur in neurotic subjects. A very large percentage of mankind is ametropic, and yet but comparatively few of these suffer in consequence. It seems that a certain amount of neurotic constitution must be present. Some ametropic patients go on for years without any trouble whatever; and then, after a severe illness or great mental or nervous strain, symptoms appear. Such cases require, after correction of the refractive error, most careful attention to the condition of the general system. In extreme cases, eye-strain may occasion not only headache, but even nausea, vomiting, vertigo and insomnia.

(*b*) **Headaches Dependent upon Disease of the Nose and Naso-Pharynx.**—The well-recognized industry of the oculist has made the existence of headaches from eye-strain a well-attested fact by all physicians. The rhinologist has not impressed the importance of his domain in this respect upon the profession. This, we think, is unfortunate, for nasal headaches are not uncommon, and, when they do occur, are apt to escape recognition. I well remember my first case of this character. There was well-marked conjunctival irritation; so I corrected the patient's refraction without benefiting him in the least. Because of a nasal obstruction, I examined his nose and found a polypus, the removal of which made a radical cure of the headache. Not only may headache arise from nasal disease, but also from certain affections of the naso-pharynx, as enlargement of the pharyngeal tonsils, follicular tonsillitis, hypertrophied tonsils, and obstruction of the Eustachian tubes. Harrison Allen has described three forms of nasal head-pains—the reflex, the neurotic, and the inflammatory—as follows: "The reflex headache is almost entirely restricted to the forehead, the temple, and the vertex. By drawing the index finger across the face from the middle of the nose to the temple, and thence, in some cases, to the parietal eminence, the patient often indicates the seat of the pain. In severe attacks, pain sometimes radiates to the vertex, and even to the nape of the neck, and then often nauseates and simulates migraine. Sometimes the point of pain is narrowed to a minute focus or spot. A very characteristic symptom is the marked increase of the headache upon the slightest exacerbation of the catarrh. A diagnostic symptom is tenderness of the inner wall of the orbit when pressed upon by the finger; or a probe passed into the nose causes an immediate access of pain when it reaches the right middle turbinated bone." The disappearance of the catarrhal reflex headache, when the nasal catarrh is cured, is the strongest proof of its nature. The neurotic nasal headache of Prof. Harrison Allen comprises cases in which highly neurotic individuals complain of pain in the throat, the ears, and back of the head, or even in the pharynx, or of various distresses about the head, as a result of a moderate degree of local nasal or pharyngeal disease. Dr. Allen further says that he has never seen catarrhal headache of inflammatory origin except in acute congestions or inflammations of the frontal sinuses; the pain is of a high grade; is, as a rule, confined to one side, and subsides after the local application of leeches.

(*c*) **Headache from Ear Disease.**—The fact that chronic aural affections other than suppurative may occasion headache is not generally recognized; yet such cases do sometimes occur. I have seen one such in which the pain was of a dull character, more or less constant, and situated just above the affected ear. There was no localized soreness, and, other than slight hardness of hearing, there were no aural symptoms. According to Dana, middle-ear disease may cause temporal and occipital pain.

(*d*) **Headache Reflex from Gastro-Intestinal Disorder.**—I have already stated that toxic headaches may occur in connection with digestive disturbances. The same is true as regards the production of reflex disturbances.

(*e*) **Headache Reflex from Disease of the Sexual Organs.**—Disease of the sexual organs in both sexes may occasion headache. In the case of the male, the difficulty is apt to be neurasthenic; in the female, however, it is a true reflex. The pains in the latter case are almost invariably situated in the vertex or in the occiput.

5. **Neuropathic Headaches.**

(*a*) **Epileptic headaches** are often spoken of, and yet I doubt if such, aside from being an occasional sequel of convulsive seizures and the still more rare occurrence of epileptic migraine, are ever seen. Dana describes what he calls an epileptic headache by saying that the pains are usually sharp and severe, often vertical or occipital, but not diffused. He says nothing about the circumstances under which the attacks appear. Gowers refers to a kind of headache which all having any experience with epilepsy have probably observed, coming on immediately after the patient awakes from the post-epileptic sleep. The pain is general, and lasts for several hours or more. Hare describes what has been called epileptiform migraine. "In these cases there is flexion of the fingers of both hands and numbness of the feet, with violent pain in the head of a hemicranial character. The tongue feels too large for the mouth, and speech is difficult. When writing is attempted (for consciousness is preserved), although words are ready to flow, only a meaningless scrawl results. The eyesight grows dim, and pain in the temples increases in violence; these symptoms being followed by vomiting and deep sleep, from which the patient awakens well. As the attacks go on, consciousness becomes slightly dimmed, but is never lost until the disease is very far advanced."

(*b*) **Hysterical headaches** characteristically assume the form known as clavus, in which the pain is limited strictly to one spot in the head,—a spot that may almost be covered by the tip of the finger. It is apt to be relieved, if not entirely driven away, by excitement or matters that appeal strongly to the patient's interest.

(*c*) **Neurasthenic headaches** are met with in persons who are "run down." The pain may be constant or deep-seated, or it may consist of certain indefinable disagreeable sensations. It is greatly increased by mental effort. Like the hysterical headache, it may disappear almost completely under excitement.

Many of the periodical headaches with which children are affected are of this type, and are due almost exclusively to the persistency with which those of a neurotic type are forced in their studies. I might say that almost all the headaches of children are due either to refractive errors or to overtaxation of the brain.

(*d*) **Migraine; Hemicrania.**—In hemicrania or migraine we have a disease which, for the present, must be regarded as a pure neurosis, occurring independently of any toxæmia or organic lesion. It is a disease of civilization, and attacks especially persons of neurotic habit or inheritance. It usually begins in early life, and exhibits a natural tendency to improve or disappear at or about middle life. Clinically, it is manifested by paroxysms of headache, associated with vaso-motor and visual phenomena. The inter-paroxysmal period, aside from the evidences of the neurotic constitution, is one of good health. Usually the paroxysms are preceded by prodromata, consisting of dull, heavy feeling, drowsiness, etc. Visual disturbances may precede the pain. They consist of hemianopsia, hemiopic visual hallucinations, or dimness of vision, or scotomata. Sometimes vertigo appears, and is very severe. Along with the visual disturbances there may be disturbed function of general sensation, as evidenced by numbness and tingling, referred to various portions of the body. It may happen that these latter symptoms are followed by motor weakness of the affected parts. The mental condition is variable—sometimes normal, sometimes depressed, and sometimes excitable. In severe cases, stupor on the one extreme and delirium on the other may appear. The headache itself usually follows closely upon the above-described sensory symptoms. The pain begins on one spot or one side, and rapidly spreads until it may involve the entire head. More than that, it may extend into the neck and arms in very severe cases. It is of agonizing character, being described by the patient as sharp, boring or throbbing. It may begin mildly and gradually increase, or it may be intense from its inception. It is greatly intensified by any sensory impression. The attacks are very prostrating, and the weakness may amount to almost absolute paralysis. Vaso-motor symptoms, in the shape of pallor of the face, retraction of the eyes, and contraction of the pupils, accompany the headache. These symptoms may be succeeded by flushing of the face, and the pupil at the same time regains its normal size. There may be œdema of the scalp and unilateral sweating. The paroxysms vary in duration from five or six hours to three days, and recur at periods ranging from two to four weeks.

(*e*) **Essential Headaches.**—Occasionally one meets with patients who are subject to headache of such a character and under such circumstances as to make it utterly impossible for the physician to determine the cause. Those I have seen have mostly been such as to make me believe that they were maintained by attention. Treatment availed nothing. Even the usual palliatives failed to palliate.

Headaches from a Topographical Standpoint.—As will be inferred from the foregoing pages, the situation of a pain in the head never has an absolute diagnostic value. Nevertheless, certain causes are more likely than others to produce headaches in certain localities; hence the situation of the pain may be regarded as having a suggestive value.

Frontal Headaches suggest

HÆMIC CAUSES.
- i. The anæmias, primary and secondary.
- ii. Toxæmia, especially lithæmia and uræmia. (Hepatic and renal toxæmias.)

NASO-PHARYNGEAL.
- i. Acute and chronic catarrhal rhinitis.
- ii. Disease of the frontal sinus.
- iii. Post-nasal adenoids.

OCULAR.
- i. Refractive errors.

- ii. Presbyopia.
- iii. Muscular asthenopia.
- iv. Glaucoma.
- v. Iritis.

ORGANIC DISEASE.

- i. Intracranial, subject to conditions already specified.
- ii. Periostitis.

Occipital Headache suggests

HÆMIC CAUSES.

- i. Toxæmia, as in rheumatism, syphilis, renal disease.

ORGANIC DISEASE.

- i. Cerebellar disease, especially tumor and abscess.
- ii. Cerebro-spinal fever.
- iii. Lesions of the basilar meninges.

NERVOUS.

- i. Neurasthenia.
- ii. Cervico-occipital neuralgia.

REFLEX.

- i. From the eyes, muscular insufficiencies.
- ii. Naso-pharyngeal diseases.

Vertical Headache suggests

Causes for the most part functional or reflex, including anæmia, utero-ovarian disease, neurasthenia, and hysteria. In the majority of cases the morbid sensations are not really pain, but paræsthesias of various kinds, including sensation of pressure, heat, etc.

Temporal Headache suggests

- i. RHEUMATISM or MYALGIA.
- ii. Disease of the TEETH.
- iii. OCULAR DISEASE, including refractive errors, glaucoma, etc.
- iv. Inflammatory affections of the MIDDLE EAR.

Pains Involving the Face.

The affections which may give rise to pain in the face include

- a. Disorders of the teeth.
- b. Facial neuritis (simple neuralgia).
- c. Tic douloureux.
- d. Disease of the ears (acute otitis media, usually).
- e. Disease of the accessory nasal sinuses.

(*a*) **Disorders of the Teeth.**—In these we have the causes of at least three-fourths of the cases of so-called facial neuralgia. While the importance of the teeth as a cause of facial pain is well recognized, the subject is often neglected clinically because the physician is usually satisfied to confine his examination of teeth to the detection of cavities. There can be no greater mistake ; for pain may originate in diseased pulp, pyorrhœa alveolaris, or, indeed, from any dental disease. We are also liable to err in supposing, because we can discover no symptoms directly referable to a diseased tooth, that therefore that tooth cannot be the cause of the neuralgic pain. Dental caries may give rise to the most severe neuralgia without any uncomfortable sensation at the site of the offending tooth. This neuralgia may frequently be ameliorated, or even temporarily cured, as I have several times observed, without treatment being directed to its exciting cause. Neuralgia from diseased teeth presents no characteristics by which it may be distinguished from neuralgia arising from other causes. It may invade any of the branches of the fifth pair. Its location does not always bear any special relation to the location of the diseased tooth. Nevertheless, when the pain is situated in the eyeball, which is not frequently the case, the offending tooth may be any of those in the upper jaw. When the pain is in the upper part of the head or in the temporal region, the diseased tooth is generally in the upper jaw and in the back part of the mouth. When the pain is referred to the ear, we look to the posterior teeth in the inferior maxilla. Gross called attention to a form of neuralgia occurring in edentulous jaws. Wedl believed that this is produced by the formation on the filaments of the torn nerves of small bodies simulating those occasionally met with on nerves after amputations. Gross himself attributed it to pressure on the minute nerves, resulting from the deposition of osseous matter within the canals. It will occasionally happen—rarely, it is true, but still sufficiently often to make it necessary for us to know of the possibility of such an occurrence—that a fang which is the cause of the neuralgia may become covered with healthy gum.

(*b*) **Facial Neuritis.**—Cases of facial neuralgia arising from inflammation of one or more branches of the trigeminus may be brought about by exposure to cold, especially to direct draughts of cold air on the face. A bilateral neuralgia of chronic duration may be produced by diabetes neuritis, though rarely. Some cases are due to herpes zoster.

Aside from neuritis, the trunk or branches of the trigeminus may be the seat of accidental lesions, many of which are difficult, if not impossible, of diagnosis. For example, severe facial neuralgia may be produced by thickening of the bony canals through which the nerve-branches pass.

(*c*) **Tic Douloureux ; Fothergill's Neuralgia ; Prosopalgia.**—In tic douloureux we have an affection occurring for the most part in persons having entered the degenerative period of life. Other than this, we know nothing concerning its etiology. The pains are of a sharp, darting character, coming, without warning, in paroxysms which last for from a few seconds to a few minutes, and then disappear as suddenly as they came.

These seizures may recur every few minutes, or they may be hours apart. The pain is usually limited to one of the branches of the trigeminus. Later other branches become sympathetically or organically affected. During the paroxysm the patient puts his hand to the affected part and exerts great pressure. Indeed, this frequently-applied pressure is often the cause of local changes in the parts, as flattening of the face and disappearance of the hair. The pain may be associated with severe local twitchings, lachrymation, coryza, or salivation. They may be excited by any movement or slight irritation of the face.

(*d*) **Diseases of the Ear.**—When pain occurs in or about the ear, it is generally symptomatic of some inflammatory condition in the middle or external ear. These inflammatory troubles are, briefly, acute catarrhal and acute purulent otitis media, and circumscribed and diffused otitis externa, and periostitis affecting the external auditory canal. In the first-named of these troubles—*acute catarrhal otitis media*—the pain is not necessarily of a severe character, neither is it always constant. It is usually aggravated at night. Sometimes the pain is of a darting character, extending from the throat into the ear, and is aggravated during the acts of coughing, sneezing, and eructation. In *acute purulent otitis media*, pain of the most severe character is often one of the earliest symptoms. It is usually continuous. Sometimes its severity is so great as to produce secondary fever and brain symptoms. It is relieved only when vent is given to the pent-up discharges by rupture of the membrana tympani. *Inflammation of the external ear* is characterized by pain, with marked sensitiveness to touch in the inflamed parts. *True earache*, in which none of the above-named lesions are present, is comparatively rare, and, when it does occur, does so as the result of disease of the teeth. It is occasionally met with in children during dentition.

Pain in the ear sometimes occurs as the result of irritation in the distribution of the second and third cervical nerves. In such cases the pain is situated in that portion of the pinna supplied by the posterior auricular nerve, namely, the posterior and lower portions. Hilton records a case of chronic earache dependent upon an enlarged gland lying close to the second cervical nerve.

(*e*) **Pain from Accessory Sinus Disease.**—The diagnosis of these cases depends upon the subjective symptoms, and not upon the pain. The reader is therefore referred to the chapter devoted to nasal symptoms and conditions (p. 322).

Pain in the Arms.

Pain in the arms may result from

1. INTRA-THORACIC LESIONS.
    a. Angina pectoris.
    b. Valvular lesions, especially aortic.

- c. Mediastinal tumors.
- d. Acute pleurisy.
- e. Aneurysm of the innominate artery.

2. SPINAL-CORD LESIONS.
   - a. Cervical hypertrophic meningitis.
   - b. Meningitis involving the cervical portion of the cord.
   - c. Locomotor ataxia.
   - d. Tumors of the cervical cord.
   - e. Morvan's disease (syringomyelia).
   - f. Vertebral disease.
3. LOCAL NERVE-LESIONS.
   - a. Cervico-brachial neuralgia.
   - b. Brachial neuritis.
   - c. Digital neuralgia.
   - d. Raynaud's disease.
4. REFLEX PAINS.
   - a. Disease of the liver.
   - b. Atony of the stomach.
   - c. Disease of the spleen.
   - d. Dental caries.

1. **Intra-Thoracic Lesions.**—(*a*) and (*b*) **Angina Pectoris and Valvular Lesions.**—The pains resulting from angina pectoris and other heart lesions were studied in the chapter on The Heart. (*Vide* p. 432.) For the present, the relation between these lesions and brachial and shoulder pains only will be considered. While the pains of the arms in angina pectoris and some other heart lesions are ofttimes atrocious, they may occasion but a moderate degree of suffering in others. The left arm is involved in the vast majority of instances. Sometimes the right arm is affected alone; and occasionally the pain involves both arms. The paroxysms are always associated with well-marked clinical phenomena indicative of the cardiac origin of the attack, especially with præcordial pain and anguish.

(*c*) **Mediastinal Tumors.**—Pain cannot be regarded as an important diagnostic symptom of mediastinal disease, because of the frequency with which it is absent until the terminal stages of the affection. Nevertheless, in many instances it is present, and is of agonizing severity. It has no settled location, as must be evident from the great variations possible in the site of the primary lesion. Sometimes, and this is the usual rule, it consists of a sense of uneasiness, discomfort, irritation, tightness, or oppression. Sometimes the original growth produces no pain, but secondary involvement of the lymphatic glands and other intra-thoracic structures leads to direct lesion of nerve-trunks or their branches. When the nerves are involved, the pains become of a shooting or lancinating type. While involving the chest-walls principally, it may extend into the neck; and, again, it may involve the brachial plexus, resulting in severe pain in the arms, with tingling and numbness extending even to the fingers.

(*d*) **Acute Pleurisy.**—With very few exceptions, the pains of pleurisy are limited to the chest. Sometimes, however, they radiate thence, and the corresponding arm may constitute a part to be thus favored.

(*e*) **Aneurysm of the Innominate Artery.**—Aneurysm of this vessel without involvement of the aortic arch is rare. In any case, however, it produces the well-known physical signs of aneurysm, as tumor, pulsation, and murmur. Pain is a prominent feature. It is not only local, but it extends up the right side of the neck and down the right arm, and is associated with numbness.

2. **Spinal-Cord Lesions.**—(*a*) **Cervical Hypertrophic Meningitis.**—The pain in these cases closely simulates that of tumor occupying the same level in the cord. The symptoms are dependent upon a hypertrophic inflammation. The lesion must necessarily have some considerable vertical extent. Pain is a prominent symptom from the very beginning, and is nearly always bilateral. Later, motor phenomena are manifested, and consist of paralysis with atrophy in the arms, and without atrophy in the legs. The sensory function of the arms is early affected.

(*b*) **Meningitis Involving the Cervical Portion of the Cord.**—The prominent sensory symptom of meningitis involving any portion of the cord is reflex pains. When, from any cause, the meninges in the cervical region are involved as part or whole of the lesion, pains are thus manifested in the arms.

(*c*) **Locomotor Ataxia.**—The characteristic shooting pains of ataxia are not uncommonly observed in the arms, though not usually until they have existed for a long time in the legs, or until the classical symptoms have become prominent. Hence their diagnosis should never present any difficulties.

(*d*) **Tumors of the Cervical Portion of the Spinal Cord.**—The symptoms closely simulate those of hypertrophic meningitis involving the same level, the more so as the growth approaches the closer to the meninges. Usually pain does not appear at first, nor is it as strictly bilateral as in hypertrophic meningitis. Later, however, it presents all the severity and extensive distribution of the other disease. The same type of paralysis,—with atrophy in the arms, without atrophy in the legs,—may prevail later. But this similarity is not likely to become a troublesome factor in differentiating, for cases of tumor are likely to terminate fatally by reason of involving important respiratory centres before these symptoms are brought about.

Inasmuch as the majority of cases of spinal-cord tumor are secondary to malignant growths elsewhere, the history of the case, taken in conjunction with the obstinacy of the pain to treatment, affords important information in establishing the diagnosis.

(*e*) **Morvan's Disease (Syringomyelia).**—Pain is an uncommon feature of Morvan's disease. When present, it is in the first portion of its course only; for later, when the whitlows appear, painlessness of the lesions constitutes the characteristic symptom upon which its diagnosis is based. The special symptoms of the disease are weakness and muscular wasting in the hands, loss of all forms of sensation, and painless whitlows on the fingers, with recurring deep ulcerations. Exceptionally, the whitlows precede the anæsthesia, in which case the lesions are painful, and continue so until sensation is destroyed.

(*f*) **Vertebral Caries.**—The pain in this condition is due to the irritation of the nerve-roots and meninges at the level of the diseased vertebræ.

3. **Local Nerve-Lesions.**—(*a*) **Cervico-Brachial Neuralgia.**—The nerves involved are the four lower cervical and the first dorsal. The areas of maximum intensity of pain are usually in the distribution of the ulnar nerve, and in the axilla and shoulder. Many conditions have undoubtedly been included under the designation of cervico-brachial neuralgia. For example, we find one that is really an occupation neurosis, in which the suffering is excited or aggravated by special movements, as in writing, and playing on the violin or piano. Sometimes the pain radiates into the chest, and thus becomes suggestive of the presence of angina pectoris. Some cases seem to originate in blows upon the shoulder. Caries of the teeth is an alleged cause. Hysteria and neurasthenia have been utilized in the explanation of still others; though, in these, careful examination usually demonstrates that the patient, instead of suffering from actual pain, has distressing sensations. Gout, rheumatism, anæmia and the neuropathic constitution have been productive of some cases.

(*b*) **Brachial Neuritis.**—As a primary affection, this occurs almost exclusively in persons of the gouty diathesis, especially in females who have passed the age of fifty. The prominent symptomatic feature is pain in the arm, associated with exquisite sensitiveness of the parts to motion or any manipulation. The pain usually follows the course of the nerves of the arm, but may be quite diffused. When the attack is a mild one, the pain, instead of being continuous, is diffused. Motor symptoms are usually absent. The principal diagnostic difficulty is encountered when the affection involves the left arm; in which case the pain may be reflected to the chest, giving rise to suspicions of angina pectoris,—suspicions which are strengthened by the presence of disturbed cardiac action. The prognosis is usually good, but the disease runs an obstinate course.

(*c*) **Digital Neuralgia.**—This is a rare affection, usually resulting from traumatism. It sometimes occurs as a reflex symptom.

(*d*) **Raynaud's Disease.**—The pain in Raynaud's disease is usually due to a local neuritis, which, however, is not a primary lesion of that disorder. It is now believed that Raynaud's disease is dependent upon vasomotor disturbance in the affected parts. It is manifested clinically by local anæmia, local congestions, and in extreme cases by local gangrene. Its etiology is practically unknown. The symptoms are rapid in their appearance, and consist of both local and constitutional phenomena, the former being the only ones present in the great majority of cases. They are best studied by describing them under three stages, namely, (1) local syncope (2) local asphyxia, and (3) local gangrene. The first stage is suddenly ushered in with numbness and various morbid sensations in the extremities, especially in the fingers and toes, and often attended with pain. Objectively the affected parts are found to be pale, bloodless, cold and shrunken. Perspiration may be either excessive or absent. The affected parts are stiff, and more or less immovable. This stage of local syncope may last from a few minutes to several days. It may constitute the entire seizure, or it may merge into the second stage, that of local asphyxia. In the latter case, the spasm of the vessels is succeeded by relaxation, and there immediately occurs an intense local engorgement. These vascular changes do not occur alike in all the affected parts. It is not unusual to note syncope in one finger, while its neighbor is in a state of asphyxia.

The stage of asphyxia may occur as the primary, and, indeed, the only one of Raynaud's disease. The affected parts assume a livid or cyanosed appearance. The tissues are swollen, and the surrounding parts become œdematous. Pain arises from the local tension, and may be quite severe. Pressure of the finger on the congested portions causes local pallor. When pressure is removed the color returns more or less rapidly according to the condition of the capillary circulation. The stage of asphyxia, if sufficiently prolonged, terminates in gangrene.

The necrotic change is first manifested, as a rule, by the formation of blebs. Sometimes, however, the affected parts simply shrivel and become black ; a line of demarcation forms, and the dead tissue is cast off. In this way the entire terminal phalanx, and even a hand or a foot, may be lost. Usually the necrosis is but superficial, involving only the skin and the immediately subjacent connective tissue. As soon as the slough is cast off, granulation takes place. The parts generally affected are the bulbs of the fingers and toes, the tips of the ears and nose. A noteworthy characteristic occurring in any situation is their symmetry. The duration of the gangrenous stage of Raynaud's disease is indefinite.

4. **Reflex Pains.**—Atony of the stomach and disease of the liver are liable to produce pain referred to the right shoulder. In the case of splenic disorder, the pain is referred to the left arm and shoulder. When dental caries produces pain in the arms, there is usually associated suffering in the neck and shoulder on the corresponding side.

Pains in the Hands.—In the majority of cases pains in the hands are dependent upon gout or rheumatism, evidence of which is found in deformities of the joints, especially in *arthritis deformans*. Sometimes they are the only clinical evidence of mild professional neuroses, especially *writers' cramp*. Sometimes pain in some particular portion of the hand, as in the thumb, is due to *teno-synovitis*.

Pains in the Lower Extremities.

Pains in the lower extremities include the following:

1. INTRA-ABDOMINAL AND INTRA-PELVIC LESIONS.
    - a. Constipation.
    - b. Pregnancy.
    - c. Pelvic tumors.
    - d. Abdominal aneurysm.
    - e. Metritis.
    - f. Salpingitis.
2. SPINAL-CORD LESIONS.
    - a. Meningitis.
    - b. Locomotor ataxia.
    - c. Tumor of the cord.
    - d. Disease of the vertebræ.
    - e. Friedreich's disease.
3. LOCAL NERVE-LESIONS.
    - a. Neuritis.
    - b. Neuroma.
4. GENERAL CONSTITUTIONAL DISEASES.
    - a. Scorbutus.
    - b. Rachitis.
    - c. Leucocythæmia.
5. VASCULAR CONDITIONS.
    - a. Phlebitis (phlegmasia alba dolens).
    - b. Erythromelalgia.
    - c. Intermittent claudication or angina cruris.
6. LOCAL TISSUE-CHANGES.
    - a. Rheumatism.
    - b. Gout.
    - c. Osteomyelitis.
    - d. Periostitis.
    - e. Growing pains.

PAINS IN THE FEET may be due to

- a. Corns and bunions.
- b. Flat-foot.
- c. Gout.
- d. Improperly-constructed or ill-fitting foot-wear.

e. Metatarsalgia.
f. Tarsalgia.
g. Rheumatism.
h. Bone-lesions.

Inasmuch as the special features characterizing the various lesions above tabulated have been previously described when speaking of pains in the arms, I shall not review the subject as thoroughly as I would were the case otherwise.

1. **Intra-Abdominal and Intra-Pelvic Lesions.**—The most severe pains in the legs arising from abdominal and pelvic lesions are due to the pressure of tumors and abscesses, and are especially liable to appear in the course of the sciatic nerve. Pressure, however, is not the only factor, for many times an extensive neuritis is set up, and this adds to the intensity of the suffering. Constipation becomes a source of suffering only in extreme cases. It acts generally by producing a toxæmia, in which case the pains are mild in degree. Sometimes, however, the accumulation of fæces in the sigmoid flexure is so great as to exert pressure upon the sciatic nerve, producing left-sided sciatica. When uterine diseases produce leg-pains, there is nearly always associated paroxysms of abdominal and pelvic pain, of which the suffering in the extremities is a prolongation.

2. **Spinal-Cord Lesions.**—There is nothing concerning the spinal pains in the lower extremities that has not already been said when speaking of pains in the arms, excepting that such pains are more frequently observed in the legs, and are of far greater severity. Especially does this refer to the peripheral pains of meningitis and locomotor ataxia.

3. **Local Nerve-Lesions.**—Of the special varieties of neuritis involving the lower extremities, two claim especial attention because of their frequency, namely, sciatica and anterior crural neuritis. **Sciatica** is unquestionably the more important of these, not only because of its frequency, but on account of the suffering it occasions. While usually referred to as a form of neuralgia, sciatica is in nearly all instances dependent upon an inflammation, rheumatic or gouty in origin, of the sciatic nerve. It occurs more frequently in males, because they are the more exposed to the exciting causes of the disease. It very rarely attacks any but adults. Some cases are produced by pressure due to faulty position. Constitutional causes, as the rheumatic and gouty diatheses and syphilis, are also important. The principal exciting causes include exposure to cold, mechanical injuries, and muscular exertion. Double sciatica is nearly always due to a constitutional poison, generally to diabetes. The principal symptom of sciatica is pain in the area of distribution of the sciatic nerve, extending ofttimes into its ultimate branches. This pain is aggravated by pressure over the nerve and by all movements which place any unusual tension upon it. The character of the pain varies in different cases, or even in the same case, from

time to time. It may be dull or sharp, tearing, burning, or boring. It is nearly always worse at night. It is quite frequently associated with a variety of morbid sensations, these including numbness and tingling in the area supplied by the sciatic nerve. Motor symptoms are of two classes. One—and that most frequently encountered—is due to the increase of pain excited by any motion. To relieve tension on the inflamed nerve, the patient walks with flexed knee. The second class is the result of weakness or actual paralysis of the muscles to which the sciatic nerve and its branches are distributed. These latter are usually limited to severe cases. Then the muscles are apt to be atrophied and flabby.

There are many conditions which have been diagnosed as sciatica, *e.g.*, hip-joint disease, disease of the spinal cord, and muscular pains in the legs; but it seems to me that even ordinary care should protect from such errors. The chief difficulty is found in the differentiation of primary from secondary cases. The persistence and agonizing severity of the pain is usually the first indication of some serious trouble hitherto unsuspected. Possibly the association of constitutional disturbance, as high fever, may suggest the possibility of the presence of some other inflammatory condition. The recognition of the cause of a secondary sciatica can only be made by thorough physical examination of the pelvic organs through the rectum or vagina, or both, and by bimanual palpation. A diagnosis of sciatic neuritis is only admissible when a careful survey of the totality of the symptoms enables us to exclude disease in all other situations.

Neuralgia of the Anterior Crural Nerve is rare as an idiopathic affection. It is usually symptomatic of morbid growths about the spine, or within the abdomen. It is sometimes due to neuritis starting in the sciatic nerve and extending to the lumbar plexus through the lumbo-sacral cord. The pain occupies the area above mentioned as being anæsthetic, but it is especially severe along the course of the nerve. The anterior crural nerve may be involved by injuries, psoas abscess, hip-joint dislocation, and disease of the vertebra.

4. **General Constitutional Diseases.**—(*a*) **Scorbutus.**—While pain is not one of the diagnostic symptoms of scurvy, it is nevertheless one which may very prominently call attention to the fact that the patient is not in good health. Especially does this remark apply to infantile scurvy, or *Barlow's disease*. It occurs mostly in the thigh, and is due to sub-periosteal extravasations of blood. The lesions start near the epiphyses and spread thence. The resultant swelling has been described as spindle-shaped. Minor degrees of severity may produce the pains in association with but slight objective local changes. The diagnosis is based upon the ordinary symptoms of scurvy, the swollen and spongy gums bleeding on slight manipulation, and the history of the case.

(*b*) **Rachitis.**—This disease is associated with a general soreness, which causes the child to suffer from any unusual manipulation or pressure. The pains are especially prominent over the epiphyses and in the legs. They not infrequently constitute an important reason for forcing the patient to remain muscularly inactive, thus leading to the impression that the patient is paralyzed (*rachitic pseudo-paraplegia*).

(*c*) **Leucocythæmia.**—Pains of a dull or boring character are sometimes observed in the tibiæ in leucocythæmia, and suggest that the bone-marrow is involved.

5. **Vascular Conditions.**—(*a*) **Phlebitis; Phlegmasia Alba Dolens.**—The pain in phlebitis is always aggravated by pressure, and is distributed along the course of the affected vessel, though more or less diffuse soreness is also present. The diagnosis, however, is based upon the associated symptoms, which include those of inflammation and venous obstruction. The affected limb is swollen and turgescent. The affected vein may show itself to inspection or palpation as a knotted cord. The surrounding tissues are brawny from infiltration, and the entire extremity is more or less œdematous. When the latter condition is at all marked, the inflamed condition of the vein is unrecognizable. Fever, usually pursuing a septic type, is present. The presence of an etiological factor capable of producing phlebitis constitutes an important point in making the diagnosis. These causes include typhoid fever, puerperal infection, influenza, diseases of the heart, and pulmonary tuberculosis.

(*b*) **Erythromelalgia.**—This is an affection the character of which is not as yet understood, and which was first described by Mitchell in 1872. It consists of severe pains in the extremities, accompanied by congestion of the affected part. It has been variously attributed to spinal, vaso-motor and neuritic changes, the probabilities in our present light being in favor of its peripheral origin. The majority of cases occur in men. The congestion of the painful part does not appear until it is made to hang down, when it at once assumes a rose-red color. The arteries of the part throb violently, and then the color becomes darker and darker, until it is finally of a purple tint. The pain is always aggravated at such times. In bad cases it may be constant; in mild ones it is only present when the part is dependent. It is worse in summer and better in winter. In some cases it prevents walking entirely. There is no diminution of sensation. The prognosis of the disease is unfavorable. According to Mitchell, bad cases do not recover. The only treatment that seems to be efficient is exsection of the nerves going to the foot. This can be done without much detriment to the patient, for they subserve unimportant motor functions.

(*c*) **Intermittent Claudication; Angina Cruris.**—"*Claudication intermittente*" was the name given this disease by Charcot when he first described it in 1858. Notwithstanding its importance and comparative frequency, it does not seem to have called forth much attention until recently, when it was thoroughly investigated by Walton, of Boston, and its cause determined to be obstruction of the arteries of the lower extremities. Typical cases are characterized by more or less frequent paroxysms of pain in the leg, involving generally the calves. Usually, the patient is attacked while standing or walking, but the pain may come on while he is lying down. Walton compares the pain in its severity to painful tic. In milder cases, the patient describes his pains as cramps. Most examples of the disease seem to go undiagnosed, being designated by physicians as sciatica, cramps, pains of flat-foot, hysteria, or the result of internal varicose veins. Walton's investigations have demonstrated that the disease is due to vascular occlusion, and that careful examination shows that the dorsalis pedis or posterior tibial arteries, or all of these vessels, are pulseless, or nearly so.

6. **Local Tissue-Changes.**—(*a*) **Rheumatism.**—The general features of rheumatic pains are found in all the disorders resulting from the rheumatic diathesis. In a measure, there is a tendency to shift from one part to another; but, while the disease remains located in one particular portion of the body, the pain is a steady, fixed one. It is associated with more or less tenderness upon pressure, and is aggravated by motion. Sometimes, after moving about for awhile, the parts become more supple, and the pain disappears.

Rheumatic pains resemble very closely those arising from sprain or other joint traumatism. Frequently the injury in these cases is so slight as to make it probable that the disproportionately great suffering is due to the awakening of dormant rheumatic disease. In acute articular, and sometimes in other varieties of rheumatism, joint changes will be evident, and establish the diagnosis.

As a rule, rheumatic pains are of gradual onset. Exceptions to this sometimes occur, and if the patient happens to be performing some labor at the time, the mistake of attributing the pain to a sprain is made.

The general tendency of rheumatic pains to aggravation during damp or cold weather should not be constituted a diagnostic symptom, for other pains exhibit the same modality, though not to the same characteristic degree.

(*b*) **Gout.**—The special locality for the gout-pain is in the ball of one or both great toes, and it is associated at some time in its course with heat, redness, and swelling of those parts. In typical gout the nature of the illness is readily recognized. But in persons who have had numerous attacks, or in those who have suffered for a prolonged period, there will occur pains which, notwithstanding the absence of the associated symptoms, must nevertheless be recognized as of gouty origin. Very often they are premonitory of a fresh paroxysm of the original disease. Sometimes they come and go without any symptomatic evidences of gout. Under the circumstances last named they partake of the nature of neuralgia or neuritis, generally in the areas supplied by the sciatic nerve. The diagnosis of such cases can never be a matter of certainty until treatment directed to the amelioration or cure of the gouty habit has been tried. Until then, it is a matter of mere inference based upon the previous history of the case.

(*c*) **Osteomyelitis.**—Osteomyelitis presents the clinical phenomena of a high grade of local inflammation with the constitutional manifestations of septic infection. Locally, we find most agonizing pain and great swelling. Associated with these are chill, high fever, delirium, and prostration. Milder cases resemble acute articular rheumatism in their symptoms. In the latter case the larger joints are usually involved, and the osteomyelitis is always multiple. Traumatism with infection is always readily traceable as the cause. The severe cases offer an unfavorable prognosis, death resulting in the course of a few days. Mild ones undergo resolution, the parts finally assuming their normal integrity.

(*d*) **Periostitis.**—Periostitis does not present the severe constitutional and local symptoms of osteomyelitis. The pain may be either dull or acute. Swelling is moderate, as are also the constitutional disturbances. The etiological factors present greater varieties than in the preceding (osteomyelitis), including not only pyogenic infection, but also syphilis and tuberculosis.

(*e*) **Growing Pains.**—The so-called "growing-pains" of children are now generally regarded as a muscular expression of mild rheumatism. In evidence of this is alleged the occurrence of chorea and cardiac lesions in many patients who have presented this symptom in the past, and the aggravation of such pains during damp weather. However this may be, there can be no doubt of the fact that the "growing pain" is not always explainable as rheumatic.

Pains in the Feet.—The diagnosis of foot-pain dependent upon corns and bunions is as easy as these lesions are evident. *Flat-foot* is responsible for severe pains of the feet which are too often attributed to rheumatism. These cases are associated with more or less spasm of muscles, produced by a dropping of the arch of the foot. Walking is very painful, and there is abduction of the foot, with subluxation of the head of the astragalus downward and forward. In some cases the loss of the arch is but slight, consisting of a mere drooping; but even in these there may be considerable pain manifested.

The influence of badly-fitting shoes in the production of pains in the feet is well known; but that badly-fitting stockings may exert a like effect does not seem to be generally known; at least, it is not recognized practically as it should be. Sometimes pains are due not so much to the so-called "fit" of the shoe as to its construction. Peculiarities in step or gait may require modifications of the ordinary foot-gear. Proper height of heel, heels made of rubber, bevelling of the sole, etc., may all have to be considered in the relief of obscure cases.

Morton's Neuralgia; Metatarsal Neuralgia.—This is a form of neuralgia the pain of which starts at the base of the fourth toe. If at all severe, it radiates into the leg. It is aggravated by pressure over the seat of pain, or by squeezing the foot. It is believed to be caused by a neuritis, the result of bruising of the nerve by the fifth bone. Some claim that it is due to a slight luxation, and still others to incipient flat-foot. It is more common in women than in men. Its diagnostic symptom is the aggravation of pain in the locality above named by lateral pressure on the foot. Mild cases yield to the adoption of a proper shoe, *i.e.*, one which permits of no lateral pressure on the foot. Severe cases sometimes require excision of the head of the fourth metatarsal bone, and even amputation of the toe.

Tarsalgia; Policemen's Disease.—This is a form of neuralgia of the foot variously attributed to flat-foot, stretching of the plantar ligaments, or contusion of the soft parts over the heel, brought on by excessive walking or standing. In the presence of a continuance of the exciting cause, treatment avails but little. Broad shoes with india-rubber heels have been recommended.

Reflex pains in the feet sometimes occur. They originate more frequently in disease in the course of the genito-urinary tract than from other causes.

Neurasthenic pains are often known to affect the feet.

Pains in the Back; Backache.

No class of pains are traceable to a greater variety of causes than are those involving the back; nor indeed is there another class of painful affections observed more frequently in practice; and we might add no class is a greater trial to the physician. Backaches may be classified as follows:

1. INTRASPINAL.
    a. Cerebro-spinal meningitis.
    b. Spinal pachymeningitis.
    c. Internal spinal meningitis.
    d. Myelitis.
    e. Syringomyelia.
    f. Tumor of the spinal cord.
    g. Hæmorrhage into the cord or meninges.
2. VERTEBRAL.
    a. Caries of the vertebræ.
    b. Cancer of the vertebræ.
    c. Arthritis deformans involving the vertebræ.
    d. Sacro-iliac disease.

3. INTRA-THORACIC.
    a. Aneurysm of aorta.
    b. Mediastinal tumors.
    c. Œsophageal disease.
4. INTRA-ABDOMINAL.
    a. Renal.
        i. Acute nephritis.
        ii. Calculus.
        iii. Renal embolism.
        iv. Hydronephrosis.
        v. Nephroptosis.
        vi. Suppurative nephritis.
        vii. Perinephritis.
        viii. Pyonephrosis.
    b. Hepatic.
        i. Cancer of liver.
        ii. Lithæmia.
        iii. Floating liver.
    c. Gastric.
        i. Ulcer of the stomach.
        ii. Atonic dyspepsia.
        iii. Acute and chronic gastritis.
        iv. Gastric neuroses.
        v. Flatulence.
    d. Pelvic.
        i. Any and all diseases of the female reproductive organs.
        ii. Cystitis.
        iii. Disease of the prostate.
        iv. Diseases of the rectum and anus.
    e. Intestinal.
        i. Indigestion.
        ii. Tympanites.
        iii. Constipation.
    f. Miscellaneous.
        i. Aneurysm of the abdominal aorta.
        ii. Appendicitis.
        iii. Abdominal tumors.
5. LOCAL.
    a. Muscular (myalgia or lumbago).
    b. Articular.
    c. Lumbar abscess.
6. CONSTITUTIONAL DISEASES.

a. Hysteria.
b. Neurasthenia.
c. Acute infectious diseases.
d. Anæmias of all kinds.
e. Sexual excesses or bad sexual hygiene.

Electro-Diagnosis.

An investigation into the reactions of the paralyzed muscles to both the galvanic and faradic currents forms a very important part of the examination of many cases; indeed, in quite a number of them it is absolutely essential to a correct conclusion. For electro-diagnostic purposes, both forms of battery are needed. The faradic machine should be one capable of furnishing a smooth, steady current, and it should be provided with a device for automatic slow interruptions of the current. The galvanic battery should be one possessing an electro-motive force of not less than fifty volts, and it must be provided with a commutator for reversing the poles, a rheostat for increasing or decreasing the strength of the current, and a milliamperemeter for measuring the amount of current in use. One can, of course, get along without either a rheostat or a milliamperemeter, just as physicians were obliged to do before these instruments were adapted to medical use; but it is decidedly inconvenient to vary the strength of a current by altering the number of cells in the circuit, to say nothing of the unpleasantness of this procedure to the patient, and the absence of a milliamperemeter makes our observations decidedly lacking in accuracy. One has only to become used to these instruments to be convinced of the inaccuracy of all his previous observations. Of the portable faradic batteries, I have used with satisfaction those manufactured by Flemming, of Philadelphia, and by the Chloride of Silver Battery Company, of Baltimore. Both instruments have given entire satisfaction. Of portable galvanic batteries, I now use only the chloride of silver battery of fifty cells, provided with a Willms's current regulator and a milliamperemeter. For office purposes, I have used for a number of years one of Flemming's wall cabinets, provided with a Dubois-Reymond coil for the faradic current, and a Massey current regulator and Flemming milliamperemeter. The current is supplied by sixty Law cells. Such an elaborate arrangement for a stationary battery is not necessary, however, as any physician with mechanical ingenuity can manufacture his own cells, and by purchasing the current regulator and the milliamperemeter can obtain as good results as he can with an apparatus of a more finished construction. The only objection that can possibly be offered to the chloride of silver battery is that of expense, and this is more apparent than real. After the first outlay the cost of maintenance is practically *nil*, while the cleanliness of the instrument and the readiness with which it may be used give it a great advantage, in the way of economy, over the many varieties of acid battery.

The application of either the faradic or the galvanic currents to healthy nerves and muscles produces muscular contractions. In the case of the nerve, the contractions are exhibited in all the muscles to which it conducts motor power. In the case of the muscles, the contractions occur only in those directly stimulated. With the faradic current the muscular contractions are of a tetanic character, and continue as long as the current passes, or until the irritability of nerves and muscles is exhausted.

The reactions to the galvanic current require longer explanation. In the first place, muscular contractions take place only at moments of making and breaking the circuit, excepting when very powerful currents are employed, when a tetanic contraction appears. Muscular contractions are more readily obtained by closing the current and applying the negative pole. This is called the cathodal closing contraction, and is designated as the CaClC. Muscular contractions also take place on breaking the circuit with the negative pole applied, but a much greater strength of current is necessary to effect this result. This is called the cathodal opening contraction, and is symbolized CaOC. The contraction at the positive pole on breaking the circuit is somewhat stronger than that on completing the same, and these are designated, respectively, anodal opening and anodal closing contractions, and their symbols are AnOC and AnClC. We have, then, as the order of irritability of muscles in response to galvanism, the following:

(1) CaClC.
(2) AnOC.
(3) AnClC.
(4) CaOC.

Disease alters the electrical reactions of paralyzed muscles, ofttimes in a characteristic manner. In certain forms of paralysis, by reason of long-continued disuse of the affected part, the electrical irritability of the paralyzed muscles becomes greatly diminished alike to both galvanic and faradic currents; in others, the phenomena known as the reaction of degeneration present themselves; and in still another class of cases no departure from the normal is observed. This last-mentioned fact needs to be emphasized, because it is a too prevalent idea that actual paralysis dependent upon organic changes must give evidence of its genuineness in the electrical reactions of the paralyzed muscles. This is a mistake; this warning should lead the examiner not to be too ready in deciding that such cases are examples of malingering.

A certain routine in the electro-diagnosis of paralytic affections is advisable to secure accuracy in results. At the outset it must be remembered that the muscles and nerves in healthy persons do not respond to electrical stimulation with equal vigor to the same strength of current. There being, therefore, no arbitrary health standard in this respect to determine what is normal for the patient under examination, one must first examine the electrical reactions of the muscles on the healthy side. If the paralysis happen to be bilateral, then certain normal muscles must be examined, their irritability compared with that of the average individual, and a standard for the case in hand determined accordingly.

When preparing the apparatus for the examination, every attention must be paid to details. The electrodes must be properly covered and moistened with warm or salt water, and the battery known to be in perfect order,—precautions altogether too frequently neglected, self-evident though they be. One electrode is used over the muscle or nerve to be tested, and the other is applied over some indifferent portion of the body, one comparatively free from motor nerves and muscles being best. The point usually selected for the latter is the sternum. This electrode should be a large flat one. For purposes of description it may be referred to as the "silent electrode." The other electrode may be in size from one-quarter of an inch to one inch in diameter; it is termed the active electrode. The greater the operator's skill, the smaller will be the active electrode with which he can work successfully.

The instruments being ready, the examiner should first test the irritability of nerves and muscles to the faradic current. Let him use the negative electrode as the active one. He must begin, as already stated, by determining the health standard. Having discovered the strength of current sufficient to produce contractions in healthy nerve or muscle, then he should study the effect of the same current applied to the corresponding point on the diseased side, and note the result. In every case it is important that he pay strict attention to the correct placing of the active electrode over the motor points of the nerve or muscle to be tested. The motor point is usually over the site of entrance of a nerve into a muscle, or at a spot where the nerve comes nearest the cutaneous surface. The accompanying cuts give the motor points of the most important nerves and muscles, and are important from a therapeutic as well as from a diagnostic standpoint in the electrical treatment of paralysis. In the case of the faradic examination it is only important to note diminished, absent, or increased muscular contractions. In the case of the galvanic current, other details of observation must be attended to. The electrodes are applied as before. The active electrode must be attached to an interrupting handle, so that the effects of sudden making and breaking of the circuit may be watched. It is best to begin with the negative as the active electrode, and the minimum current capable of producing muscular contractions at making and breaking of the circuit determined. The character of the muscular contractions must also be observed. In health, the muscles respond with a short, quick jerk. Having determined the electro-muscular irritability to the negative pole, the same should be investigated with the positive pole, and the results noted. Departures from the galvanic reactions in health may be noted in several directions. In the first place, the irritability of the nerves may be diminished or increased in direct proportion to the alterations in faradic contractility. The muscles themselves may be similarly affected. These changes are called quantitative alterations. In the second place, we may find important changes in the character of the reactions. These are known as qualitative alterations. The galvanic irritability of the muscles may be increased, while the faradic is diminished, or even destroyed. The resulting muscular contractions are of a slow, wavy character, and not short and quick, as in health; and there are important changes in the polar reactions. Thus, instead of the cathodal closing contraction being the most marked, we find the anodal opening or anodal closing contraction the strongest. For purposes of record we may use the symbols already given, and designate their relations by the use of the crescendo, diminuendo, or equality signs. Thus CaClC > AnOC would indicate cathodal closing contraction stronger than anodal opening contraction. Reversing the sign, making it a crescendo, in other words, and we indicate the anodal opening contraction as the stronger.

While definite rules respecting the alterations in the polar reactions have been formulated, they are by no means reliable clinically, as their significance can hardly be said to have as yet been determined. These qualitative alterations in galvanic response, together with diminished or absent faradic irritability, constitute the reaction of degeneration. For all practical purposes in actual practice it is necessary only to discover the diminished faradic and increased galvanic response and the alterations in the character of the muscular contractions. In many cases in which the faradic irritability is so slightly altered as to be almost imperceptible, the altered galvanic response becomes of the greatest importance.

Departures from the characteristic reaction of degeneration as above described may be observed under certain circumstances. One is known as the chronic form, and is characterized by diminution of faradic and galvanic irritability of nerves and muscles alike, with the qualitative galvanic changes above described. The other is called the "mixed form." It is characterized by slight or marked changes in the muscle irritability, while the nerves react normally, or nearly so. The alterations in the muscle reactions consist of increased or diminished galvanic irritability and changed polar reactions. This condition is observed in cases in which some of the muscular fibres are degenerated, while others are normal, as in some cases of peripheral paralysis and progressive muscular atrophy.

The reaction of degeneration in its typical form has a well-defined course. At about a period ranging from two to three days to as many weeks, there appears the diminished faradic irritability, which progresses more or less rapidly to complete destruction according to the severity of the case. At the same time the galvanic irritability of the nerves diminishes. In the course of from one to two weeks the galvanic irritability of the muscles increases as above stated, and reaches its height in about a month, when it, too, begins to diminish. In marked cases it may go on to complete abolition. At periods ranging from two to six months restoration takes place, in a measure, in some cases,—this, of course, being governed by the severity of the primary lesion.

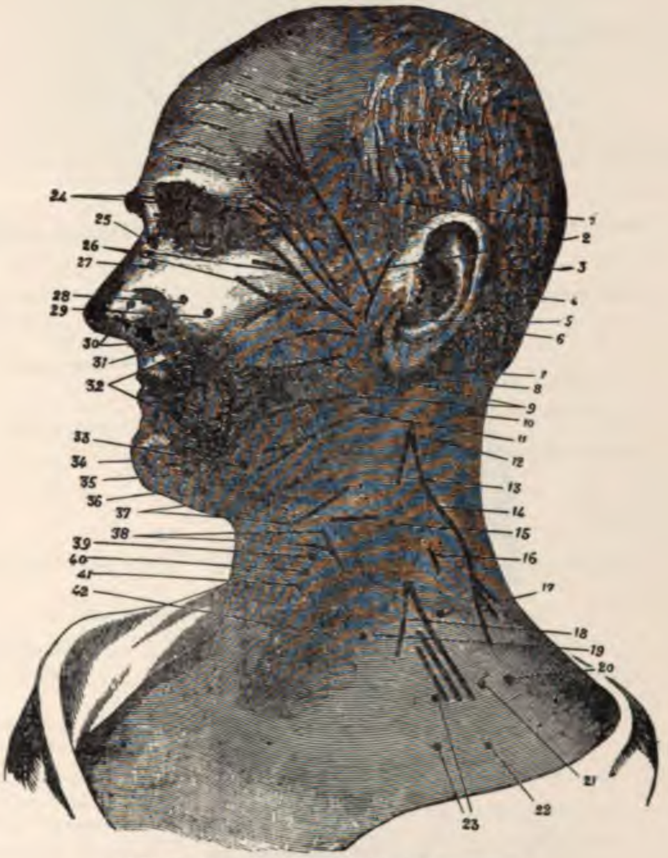
The reaction of degeneration is of diagnostic value, as it indicates certainly a lesion affecting a motor nerve or its nuclear cells. Hence we find it present in acute poliomyelitis, nerve injuries and inflammations, and in degenerations of the nuclei of the cranial nerves. It is found in its chronic form in progressive muscular atrophy and amyotrophic lateral sclerosis.

Simple diminished electrical irritability without qualitative changes is found in cerebral palsies after the paralysis has lasted three or four months, in certain spinal-cord diseases, as amyotrophic lateral sclerosis, and after slight nerve injuries and inflammations, and in certain muscular conditions, as arthritic muscular atrophy, atrophy from disuse, and myopathic atrophy.

Increased electrical irritability without qualitative changes is observed in the early stages of hemiplegia and of acute myelitis, progressive muscular atrophy, chorea, locomotor ataxia, and Thomsen's disease. In the last-named a peculiar condition is sometimes observed. It is called the myotonic reaction, and it consists of increased faradic contractility with prolonged contraction after the current ceases, and in excessive galvanic irritability when the circuit is closed with sluggish and wavy contractions.

The many other symptoms, significant of nervous disease and nervous complications of other disorders, might well be considered with advantage at this time. Inasmuch, however, as they will be fully treated in other portions of this work, their study here is unnecessary.

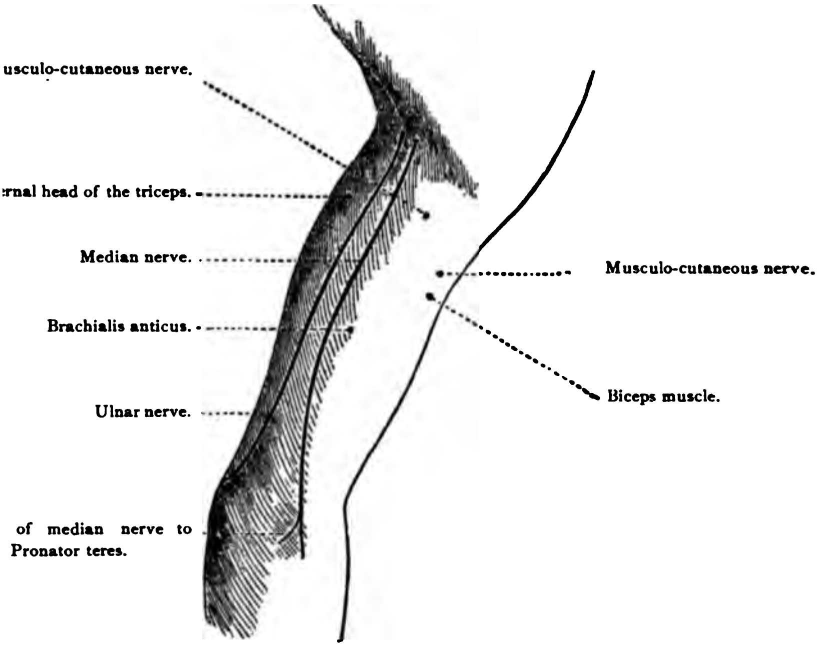
FIG. 200.



Motor points of head and neck (Ziemssen).

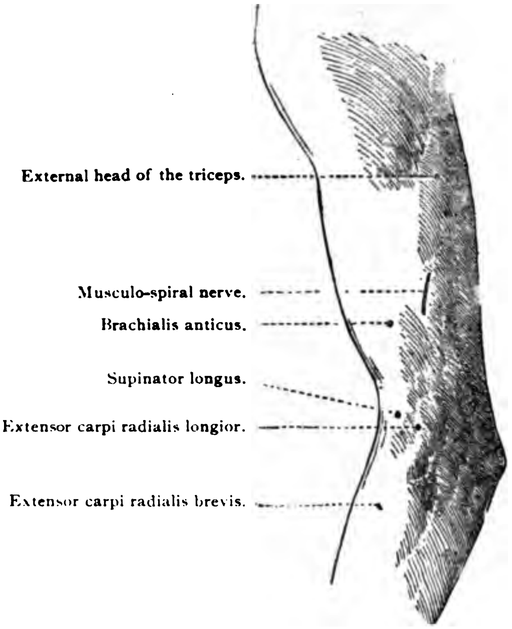
1. Frontalis muscles.
2. Attrahens and attollens auriculam muscles.
3. Retrahens and attollens auriculam muscles.
4. Occipitalis muscle.
5. Facial nerve.
6. Posterior auricular branch of facial nerve.
7. Stylohyoid muscle.
8. Digastric muscle.
9. Buccal branch of facial nerve.
10. Splenius capitis muscle.
11. Subcutaneous branches of inferior maxillary nerve.
12. External branch of spinal accessory nerve.
13. Sterno-mastoid muscle.
14. Cucullaris muscle.
15. Sterno-mastoid muscle.
16. Levator angulæ scapulæ muscle.
17. Posterior thoracic nerve.
18. Phrenic nerve.
19. Omohyoid muscle.
20. Nerve to serratus magnus muscle.
21. Axillary nerve.
22. Branch of brachial plexus (musculo-cutaneous and part of median).
23. Anterior thoracic nerve (pectoral muscles).
24. Corrugator supercilii muscles.
25. Compressor nasi and pyramidalis nasi muscles.
26. Orbicularis palpebrarum muscle.
27. Levator labii superioris alæque nasi muscle.
28. Levator labii superior muscle.
29. Zygomaticus minor muscles.
30. Dilator naris.
31. Zygomaticus major.
32. Orbicularis oris.
33. Branch to triangularis and levator menti muscles.
34. Levator menti muscle.
35. Quadratus menti muscle.
36. Triangularis menti muscles.
37. Cervical branch of facial nerve.
38. Branch to platysma muscle.
39. Sternohyoid muscle.
40. Omohyoid muscle.
41. Sternothyroid muscle.
42. Sternohyoid muscle.

FIG. 201.



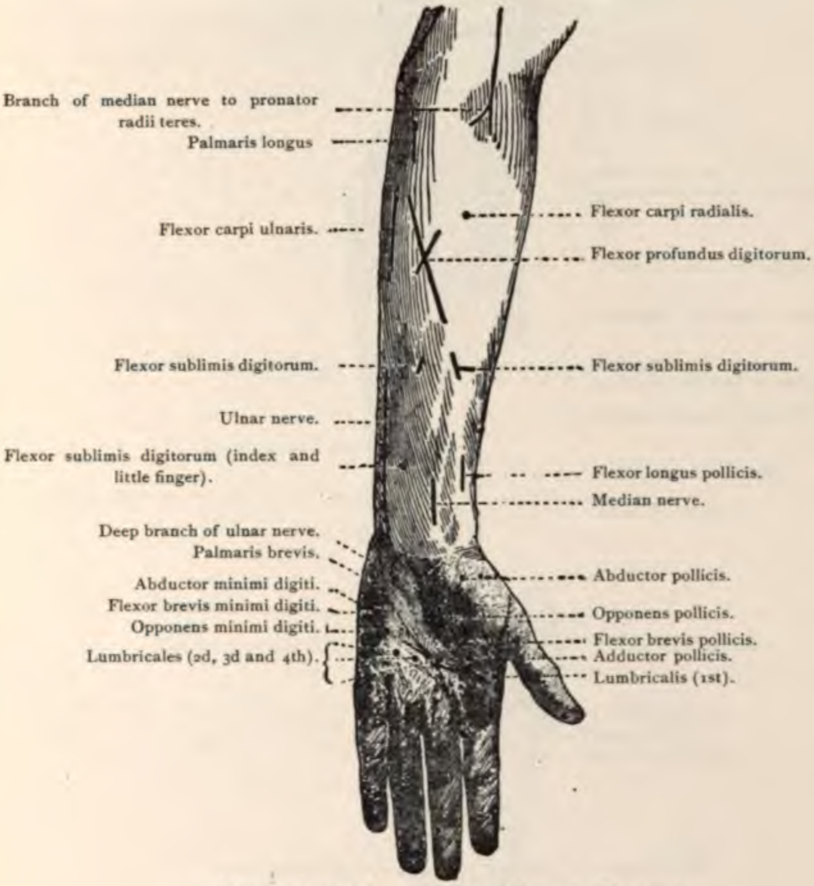
Motor points of the arm (Ziemssen).

FIG. 202.


External head of the triceps.
Musculo-spiral nerve.
Brachialis anticus.
Supinator longus.
Extensor carpi radialis longior.
Extensor carpi radialis brevis.

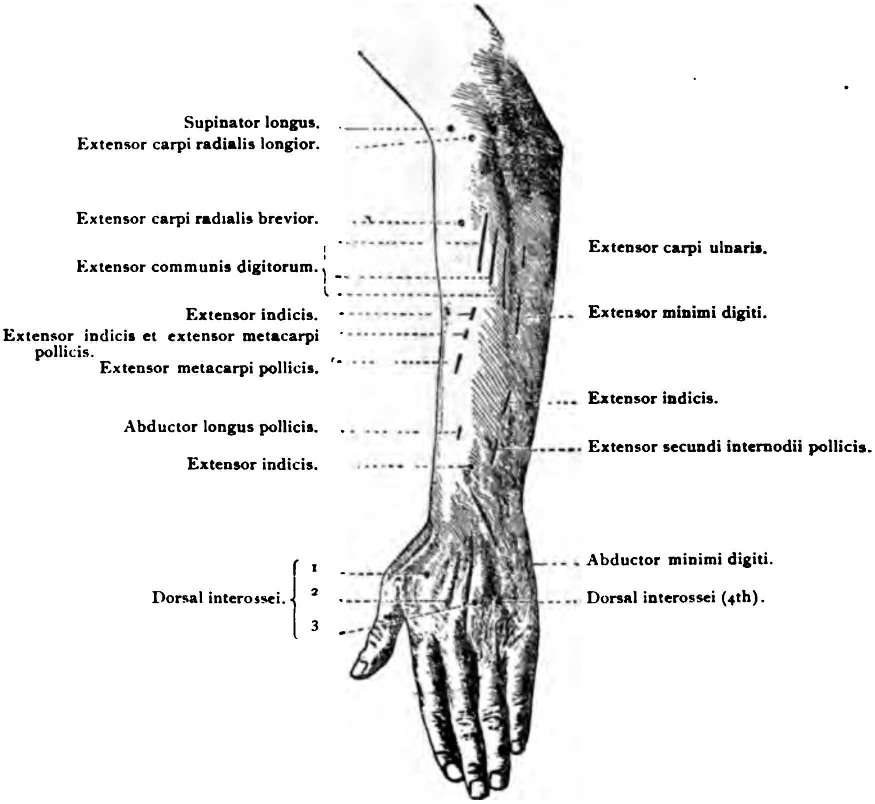
Motor points of posterior surface of the arm (Ziemssen).

FIG. 203.



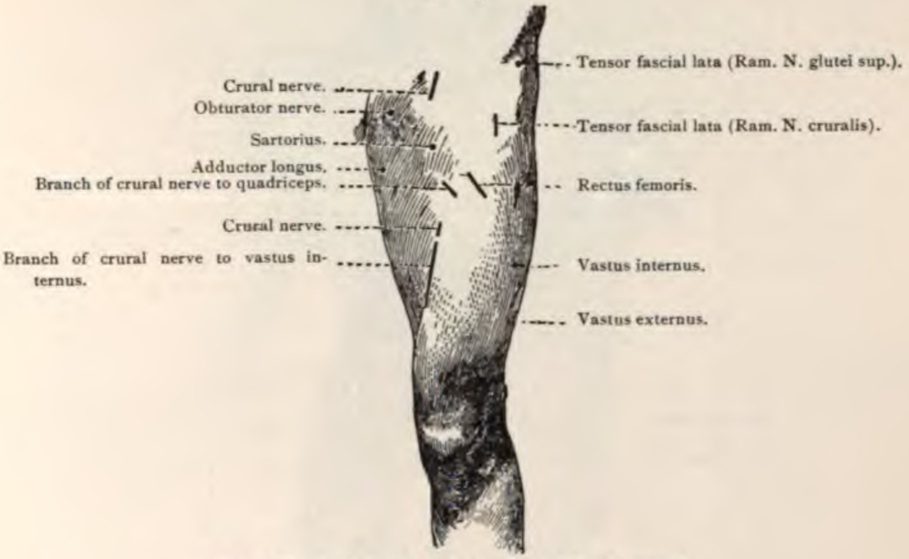
Motor points of flexor aspect of forearm (Ziemssen).

FIG. 204.


Supinator longus.
Extensor carpi radialis longior.
Extensor carpi radialis brevior.
Extensor communis digitorum.
Extensor indicis.
Extensor indicis et extensor metacarpi pollicis.
Extensor metacarpi pollicis.
Abductor longus pollicis.
Extensor indicis.
Dorsal interossei.
1
2
3
Extensor carpi ulnaris.
Extensor minimi digiti.
Extensor indicis.
Extensor secundi internodii pollicis.
Abductor minimi digiti.
Dorsal interossei (4th).

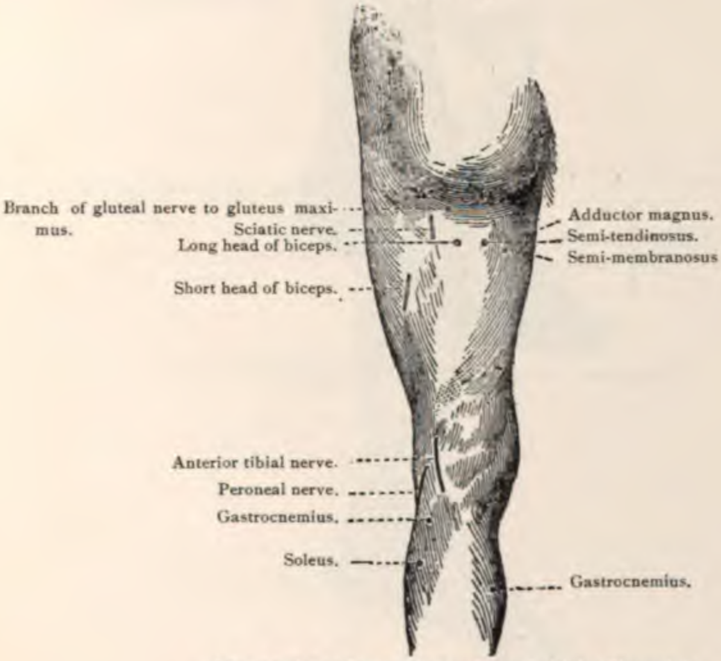
Motor points of extensor aspect of forearm (Ziemssen).

FIG. 205.


Crural nerve.
Obturator nerve.
Sartorius.
Adductor longus.
Branch of crural nerve to quadriceps.
Crural nerve.
Branch of crural nerve to vastus internus.
Tensor fascial lata (Ram. N. glutei sup.).
Tensor fascial lata (Ram. N. cruralis).
Rectus femoris.
Vastus internus.
Vastus externus.

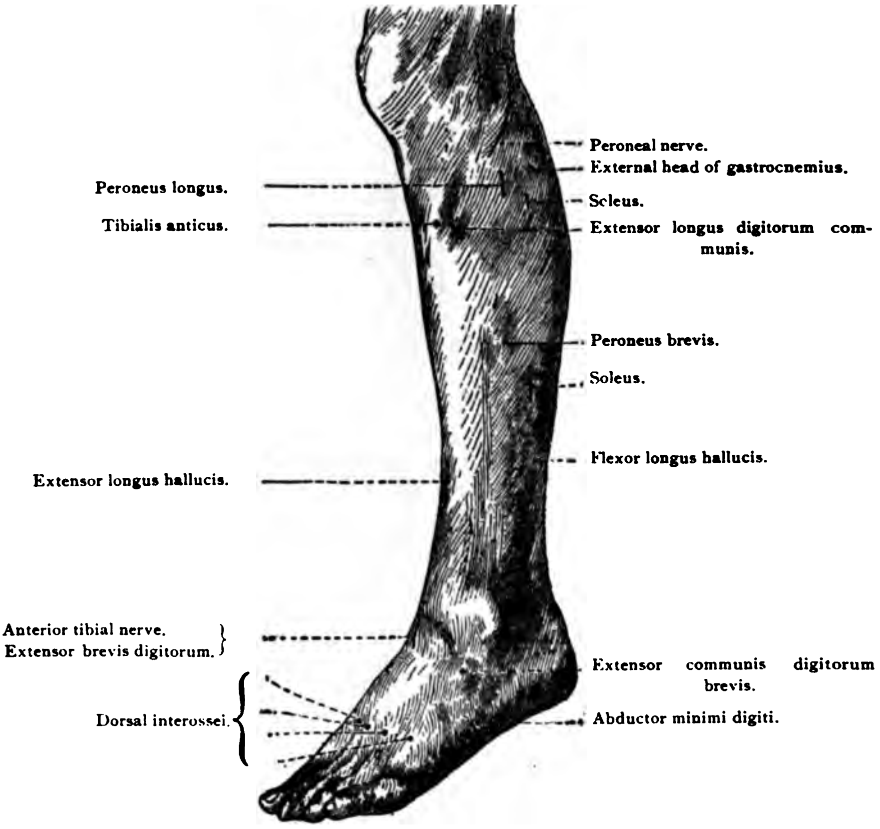
Motor points of anterior surface of thigh (Ziemssen).

FIG. 206.



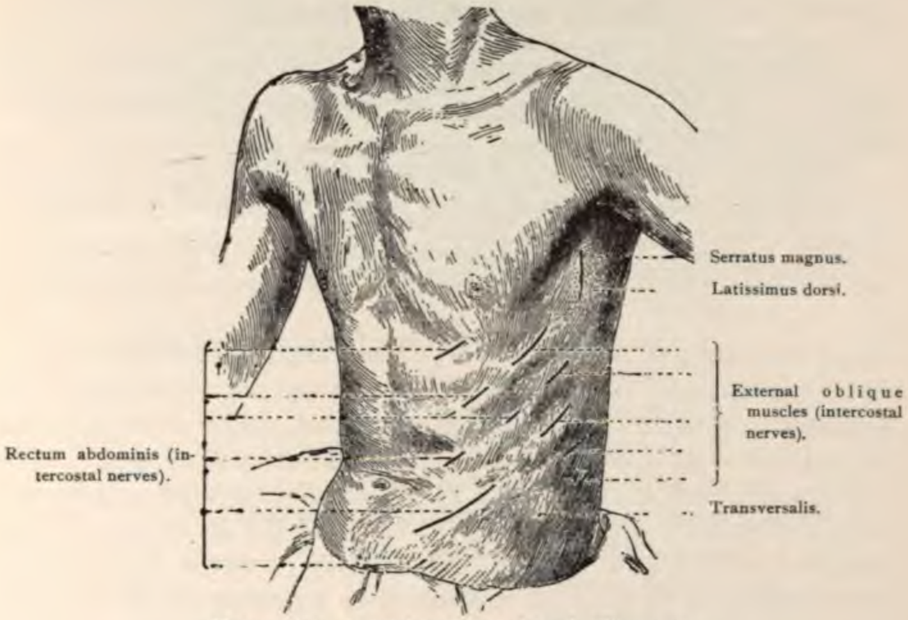
Motor points of posterior surface of thigh (Ziemssen).

FIG. 207.



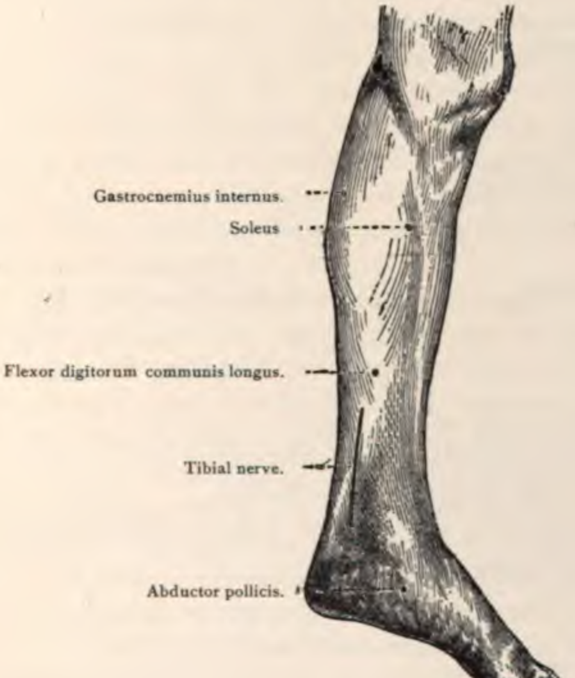
Motor points of outer surface of leg (Ziemssen).

FIG. 208.


Serratus magnus.
Latissimus dorsi.
External oblique muscles (intercostal nerves).
Rectum abdominis (intercostal nerves).
Transversalis.

Motor points of anterior surface of trunk (Ziemssen).

FIG. 209.



Motor points of inner surface of leg (Ziemssen).

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y. S

While the clinical data of each of the above affections are all-sufficient to establish a diagnosis in every case of coma, were all such data obtainable or at hand, it is a fact that the circumstances under which patients with coma are frequently seen by the physician make the diagnosis of this condition one of the most difficult problems with which the practitioner has to deal. In many of the mysterious cases, possibly the majority, a diagnosis is out of the question, simply because the patient is unable to give a history of the case. Provisional diagnoses made with great confidence during life are frequently refuted at the autopsy, showing the danger of reaching conclusions without absolute certainty of being in possession of all the data.

(*a*) **Cerebral Traumatism.**—This subject has already been considered in part, when treating of the cerebral palsies of sudden onset. The data upon which we are accustomed to rely as demonstrating the traumatic origin of a given case of coma are mainly the objective signs of injury, as contusions or lacerated wounds of the scalp, bleeding from the ear, etc., and the absence of a history—when the patient is well known—of other possible causes of coma. But the fact that an individual is liable to become comatose without warning is no evidence of the non-traumatic origin of the attack, for such individuals—epileptics, for example—are liable to fall and sustain head injuries. Such positive evidence as the presence of injuries of the soft parts should outweigh all other facts or absence of facts, and lead to the performance of an exploratory incision of the scalp to determine the integrity of the skull. Further than this one is not justified in proceeding, excepting other positive diagnostic symptoms are present. Of the latter class of symptoms, none is of greater importance than the presence of a lucid interval between the reception of the injury and the onset of unconsciousness. This interval may be but a few seconds, or it may be many hours; but when present it is diagnostic of meningeal hæmorrhage, and calls for trephining.

(*b*) **Idiopathic Cerebral Hæmorrhage.**—The majority of cases of cerebral apoplexy are comparatively easy of diagnosis. (*Vide* p. 705.) But when the patient is found unconscious under suspicious or doubtful circumstances, and the intracerebral hæmorrhage has invaded an unusual locality, the problem is difficult if not impossible of solution. Given a patient found unconscious, a diagnosis of apoplexy will be made when the pulse is of high tension, the coma is attended by stertorous breathing, with flapping of one cheek and asymmetry of the face, and restlessness of one side of the body only, showing that the other side is incapable of motion. Two symptoms or conditions not uncommonly found in the unconscious may mislead the physician from a correct diagnosis based upon the above-mentioned data. They are the odor of alcohol upon the breath and the presence of albuminuria. The odor of the breath should count for little, because many apoplectics are seized after an evening spent in carousing, and persons long habituated to large doses of alcoholic liquors are liable to vascular degeneration. The presence of albuminuria should not necessarily suggest a diagnosis of uræmia, for diseased kidneys are of common occurrence in apoplectic patients.

(*c*) **Cerebral Embolism and Thrombosis.**—The onset of unconsciousness in cerebral thrombosis is without preliminary symptoms. The diagnosis is based upon the recognition of cardiac murmur and the absence of conditions liable to produce intracerebral hæmorrhage, as degenerated arteries, renal diseases and syphilis, or thrombosis, as syphilis and old age.

(*d*) **Cerebral Tumor.**—Unconsciousness may supervene as a paroxysmal symptom in the course of a case of cerebral tumor, or it may constitute one of the terminal phenomena. Occurring as a paroxysmal symptom, it may be in association with or a sequence of a convulsive seizure; or, as in some cases of epilepsy, it may constitute the only feature of the attack. The appearance of coma as a terminal symptom is so gradual that the case, under such circumstances, is hardly likely to come under the care of a physician who is not already fully conversant with the patient's condition, so that diagnostic difficulties are not likely to arise. As to the paroxysmal coma, it is difficult to lay down any hard and fast rules to demonstrate that the origin of the trouble is a tumor, unless the full history of the case is obtainable. The discovery of a double optic neuritis would, of course, count for much; but then the ophthalmoscopic examination of comatose patients is not an easy matter.

(*e*) **Cerebral Abscess.**—The diagnosis of coma due to cerebral abscess is based entirely upon the presence of signs of conditions liable to produce cerebral suppuration. Thus, a patient was found by the police patrol unconscious in his room, where it was believed he had been for at least two days. He was brought to the Hahnemann Hospital. He had a very offensive otorrhœa, which led to my diagnosis of cerebral abscess. The same diagnosis would be justified in the case of a patient found unconscious with an infected wound of the scalp.

(*f*) **Cerebral Meningitis.**—Ordinarily, the coma or stupor of meningitis of any variety comes on only after several days of suffering sufficient to keep the patient confined to his bed. Usually, too, it is preceded by more or less delirium. Exceptional cases may occur in which the above does not hold true, as in a case already reported by me in which a man became unconscious while on a train between New York and Philadelphia. On his arrival he was removed to the Hahnemann Hospital, where he died. The autopsy showed a tubercular meningitis. Such cases present a clinical picture so far removed from that which we ordinarily expect in this disease that it is doubtful if we can lay down any rules for their correct diag-

(*g*) **General Paralysis of the Insane.**—The course of this disease is sometimes marked by attacks of coma, which were described by Charcot, and referred to by him as apoplectiform seizures. Some of these are almost exact clinical counterparts of true apoplectic seizures, and are followed by hemiplegia, which continues for a few days. Some end fatally. Sometimes the attack comes on gradually in the course of one twenty-four hours, to depart as gradually during the next, leaving the patient with a certain amount of clumsiness or motor incoördination. The course of general paralysis of the insane may also be interrupted by attacks of syncope, which last but a few minutes.

(*h*) **Epilepsy.**—In the majority of cases of epilepsy the coma is of short duration. Most of the cases, moreover, give some evidence of the preceding convulsion. Examination of the mouth will disclose the presence of old cicatrices on the tongue, showing that that organ has been severely bitten in the past.

(*i*) **Narcotic Poisons**: i. **Alcohol.**—There is too strong a tendency in police and hospital circles to regard too many cases of stupor as examples of acute alcoholism, especially if it so happens that the patient's breath gives forth an odor of liquor. Such snap diagnoses have been fraught with serious consequences in the past. Of course the odor of alcohol on the breath is an important positive sign; but it is not pathognomonic. It becomes doubly important, however, in the absence of signs and symptoms indicative of other possible causes of coma. The unconsciousness of drunkenness is rarely so profound that the patient cannot be aroused with a little effort. It also corrects itself in a few hours, if the patient is kept from further indulgence. Very exceptionally, owing to personal susceptibility or to the taking of abnormally large quantities, the coma of acute alcoholism is profound. The face is pale; the pupils are generally dilated and inactive to light; the pulse is very weak, and the skin cold. Death takes place through paralysis of the respiratory centres. These cases are so rare, however, that they need not interfere with the general diagnostic principles involved.

ii. **Opium and its Alkaloids.**—When the preparation taken happens to be laudanum, the odor of the drug on the breath constitutes an important indication, which is absent, however, in the case of poisoning with the other opium preparations. Symptoms appear within an hour after the poison has been taken. After a brief period of excitement the patient becomes somnolent, and quickly passes into a condition of absolute stupor, with complete muscular relaxation all over the body. The skin is bathed in a profuse perspiration. The pupils are contracted to a minimum size, and are irresponsive to light. This last is a very important symptom. The pulse is at first full and strong, later becoming weak, slow and irregular. Respirations are slow and stertorous, later associated with large, moist râles. In fatal cases death usually takes place within twelve to eighteen hours after the exhibition of the poison.

iii. **Illuminating-Gas Poisoning.**—The diagnosis of these cases is clear, because the patient is always found unconscious, with the odor of gas permeating the room, and the gas freely escaping from the burner.

iv. **Chloral-Hydrate Poisoning.**—The symptoms of chloral poisoning present a most remarkable resemblance to those of opium, the main distinction being the condition of the pupils. In the case of chloral poisoning they are moderately dilated ; or, if they should be contracted, that contraction is but moderate, and disappears if the patient is aroused. The odor of the drug may be of some service when a sufficiently large quantity has been taken.

(*j*) **Sunstroke.**—Weather conditions, with high fever (hyperpyrexia), make the diagnosis of sunstroke easy, especially as circumstances are such that a complete history of the case is usually forthcoming.

In heat prostration the unconsciousness is rarely profound. There is a restlessness, with cold, clammy surface, weak pulse, and even delirium in most of the cases.

(*k*) **Uræmia.**—As to what should be called uræmic coma is, in the opinion of the writer, a debatable question. Custom sanctions the application of the term to all cases of coma attended by undoubted evidences of advanced renal disease, *i.e.*, by albuminuria, greatly diminished urine, if not complete suppression, diminished urea excretion, and tube-casts. As additional signs confirmatory of the diagnosis are œdema of various portions of the body, and the usual cardio-vascular conditions attendant upon chronic nephritis. But numerous cases are observed in which the urea elimination does not seem to be sufficiently defective to warrant the production of the profound coma.

It is usually taught that the coma of uræmia is of slow onset ; and yet those who have had hospital experience have seen numerous instances in which cases permitting of no other diagnosis have been picked up unconscious on the streets. The coma is also alleged to be a sequence of convulsive symptoms ; and so it is in many instances, but not so frequently as to make the association an important matter in the making of the diagnosis.

(*l*) **Diabetic Coma.**—A very large proportion of cases of diabetics terminate fatally in coma, which for the most part is of rapid onset. It is especially liable to occur in comparatively young subjects. It is frequently associated with peculiar disturbances of respiration, which has been described as a dyspnœa. The respirations are much deeper, and are more forcible than normal, and somewhat increased as regards their frequency. The pulse is quite rapid—120 to 140—and the temperature, if disturbed, is subnormal. The causes leading up to this remarkable coma remain unknown as yet; though there is a growing conviction that the condition is one of acid-intoxication. By some it is attributed to a too rigid adherence to a non-starchy diet. The discovery of acetone and diacetic acid and sugar in the urine should lead to a correct diagnostic conclusion.

(*m*) **Syphilis.**—Syphilis may be the cause of coma through the medium of one of the special pathological changes which it may produce, as gummatous growths, meningitis, etc. There is, in addition, a special form of coma, to which Althaus has assigned the name "*syphilitic coma.*" Cases of this character are rare. The premonitory symptoms are headache, mental confusion, drowsiness, indistinctness of speech, numbness, and some slight loss of power. The symptoms of the attack set in during sleep, the patient being found in bed in the morning, unconscious. In some cases in which the coma is not profound the patient may be aroused sufficiently to answer questions, but he immediately relapses into his former condition. The tongue is dry, and is protruded with difficulty on command; swallowing is not readily performed. The eyeballs are sunken in their sockets, and there is apparently a slight outward deviation of the eyes, which is in direct proportion to the intensity of the coma. Reflex excitability everywhere is diminished. There is incontinence of urine and fæces. The pulse is usually quite slow, but its character is variable. Respiration is slow and shallow, and the temperature is below normal.

(*n*) **Malaria.**—Coma has a place in the symptomatology of malarial fevers only in those cases of ordinary intermittents which have been neglected or permitted to continue under bad treatment, and in the comatose variety of pernicious fever. In the latter case the disease may be ushered in by a suddenly appearing coma, in which the patient dies within twelve to twenty-four hours. As variations from this mode of onset, we may have the initial phenomena consisting of a chill, or there may be several paroxysms of intermittent fever before the coma has asserted itself. The patient may come out of the first attack of coma only to go into another, which proves fatal. The diagnosis of these cases rests upon a knowledge of climatic conditions and the symptoms above noted, and the examination of the blood.

(*o*) **Cholæmia.**—In liver diseases attended by jaundice there is not infrequently a profound auto-intoxication, the precise nature of which does not seem to have been positively determined. The two theories advanced for the explanation of the condition are as follows: (1) That the liver fails to separate from the blood the substances that go to make up the bile, and (2) the entrance of the bile into the blood. The first of these seems to be the more acceptable, for the phenomena of intoxication are not necessarily proportionate to the degree of discoloration of the tissues by the bile. The clinical phenomena constituting cholæmia include slowness of the pulse, hæmorrhagic tendency, numerous nervous symptoms, of which delirium, coma, convulsions, tremor and paralysis are the chief, and hemaralopia. The nature of the coma in these cases is easily discerned, because of the unequivocal character of the associated symptoms.

(*p*) **Acute Yellow Atrophy of the Liver.**—The coma attendant upon this disease is due to the associated cholæmia. It usually appears after the disease has continued for several days, and grows deeper until death.

(*q*) **Acute Infectious Diseases of the Foudroyant Type.**—The acute infectious diseases which may present coma as an early symptom by reason of the severity of the toxæmia include scarlatina, typhus, cerebrospinal fever, yellow fever, variola, æstivo-autumnal fever, and numerous others. Their diagnosis rests almost exclusively upon a knowledge of epidemic and climatic conditions.

(*r*) **Exhaustion in the Course of Acute Diseases.**—Fatal cases of any of the acute or chronic diseases may present coma as one of their terminal phenomena. No diagnostic difficulties are involved in these cases, because it is the almost invariable rule that a correct diagnosis has been made long before this unfortunate stage has been reached.

(*s*) **Cerebral Malnutrition from Weakened Heart and Degenerated Arteries in Old Age.**—Drowsiness is a common condition in the aged. It is usually dependent upon malnutrition of the brain due to defective blood-supply (virtual anæmia), the result of the degenerated blood-vessels and the weakened heart. Drowsiness is not, however, the only symptom that may result from this cause. For example, arteriosclerosis is a frequent cause of vertigo.

(*t*) **Dyspepsia.**—After a full meal, drowsiness is not an uncommon symptom. It is brought about by two agencies: (1) An auto-intoxication; and (2) by dilatation of the abdominal blood-vessels withdrawing the blood from other portions of the body, the brain included. The beneficial effect of systematic exercise in oxidizing the waste materials in the cases in which auto-intoxication is an important element is well-known.

(*u*) **Obesity.**—The drowsiness of obese subjects is dependent upon auto-intoxication, mainly, though it is due in part to the dilatation of the abdominal muscles and the weakened cardiac muscle.

(*v*) **Narcolepsy; the African Sleeping Sickness.**—This is a disease observed among the negroes of the west coast of Africa, among the inhabitants of the island of Martinique, and in some other localities. It is characterized by a gradually deepening coma attended by emaciation, and ending in death. Associated symptoms include ptosis, puffiness of the face, enlargement of the cervical glands, and tremor. Its pathology and etiology are unknown.

(*w*) **The Initial Stages of Some Cases of Insanity.**—The usual disorder of sleep in cases of insanity is sleeplessness. Exceptionally morbid somnolence is observed; or, the patient may exhibit drowsiness; but as soon as he makes the attempt to go to sleep, the ability or inclination to do so disappears. The majority of the cases presenting this symptom may be characterized as border-line cases; their victims are simply neurotics.

(*x*) **Hysteria.**—The condition presented by a patient in hysterical coma is difficult to describe; and yet the manifestations are such that they appeal at once to the experienced. Nearly always there is a history of good bodily health with an unstable nervous organization, and the occurrence of some emotional disturbance just preceding the paroxysm. The coma itself is never profound, but is disturbed by laughing, crying, coördinate muscular movements (hysteroid spasms), and talking. The facial expression is different from that observed in cases of unconsciousness due to all other causes. The patient is usually a young woman. She may present the appearances usually observed in hysterical subjects. The mannerisms of the family afford some clue to the nature of the case, for it is well known that hysteria finds its most important cause, aside from the neuropathic constitution, in education and environment. In addition to the above positive signs, we have, as aids to diagnosis, the absence of signs and symptoms indicative of the other causes of coma.

(*y*) **Syncope.**—Fainting only takes place in those predisposed by reason of bad health or the neuropathic constitution. In the latter cases some emotional influence constitutes the exciting cause. Such cases are closely akin to the hysterical. Syncopal attacks are characterized by pallor of the face, coolness of the surface, and weak pulse and respiration. When the paroxysm is dependent upon absolute cardiac weakness, the pallor is associated with cyanosis and some stertor.

Delirium and Allied States.

Delirium is a condition occurring in the course of a multitude of diseases characterized by incoherence of thought, speech, and action. It is in no sense a disease.

It is referred to as **low** and **active.** The delirious patient exhibits his mental state by incoherent speech, restless movements, and more or less complete obliviousness of all surroundings. In **low** or **muttering delirium** the patient lies quietly in bed, mumbling more or less unintelligently, his words being addressed to imaginary persons, and relating entirely to imaginary affairs. In **active** delirium the patient tries to escape from the bed or from his room, struggles violently with those who attempt to restrain him, and is very noisy.

Delirium may be observed in the following general conditions:

a. Fevers.
b. Toxæmia.

c. Asthenia.
d. Neuropathic.
e. Certain poisons.

The personal factor, *i.e.*, the neuropathic constitution of the patient, has in most cases a very important bearing in the production of delirium in response to the exciting cause, as demonstrated by the readiness with which certain individuals become "flighty" to very slight influences. Children in particular are thus affected. Indeed, we expect the latter class of patients to become somewhat delirious, even with their slighter ailments.

(*a*) **Fevers** and (*b*) **Toxæmiæ.**—In the majority of cases of febrile delirium toxæmia plays some part in the production of this symptom, and fever is an important factor in the production of some of the cases of toxæmic delirium. Of the fevers which are especially important in this respect, typhoid fever, scarlatina, intermittent and other malarial fevers, yellow fever, influenza and the septic infections are especially important. In *typhoid fever*, the delirium does not usually assert itself until the second or third week in the disease. Occurring thus, it may be the result of the long-continued pyrexia, the asthenia, or the toxæmia, singly or in combination. In most instances it is of the low, muttering kind. In *scarlatina*, delirium is a prominent and an early symptom. This is accounted for by the tender years of the patient and the high temperature. It is greatly intensified by the toxæmia. In the *malarial fevers*, the delirium is brought about by the pyrexia and the susceptibility of the patient, when it is an early phenomenon. Occurring late, it is an expression of the systemic poisoning and the asthenia. In *yellow fever*, delirium is of common occurrence, so that it may be regarded as one of the regular symptoms of the disorder. The delirium of *influenza* is a most irregular phenomenon. It is commonly absent in all stages of the disease; but sometimes the attack is ushered in by flightiness, which may lead to considerable apprehension on the part of attendants until its true nature is recognized. Under such circumstances, it and the fever may constitute the only evidences of ill-health. It may be of the active, wild variety. The delirium of *septic fever* is low and muttering, and is dependent almost entirely upon the toxæmia. Other diseases than those above quoted may be attended by delirium, but they are too numerous to require mention in this place.

It is difficult to define the position of the delirium which accompanies pneumonia, pericarditis, pleurisy, peritonitis, and rheumatism. No doubt, in these cases, we have a multiplicity of causes at work. In pneumonia, the pyrexia and the toxæmia are important; but it is also probable that in some instances, at least, meningeal infection is a factor. The delirium of croupous pneumonia is ofttimes such a prominent feature of the illness that it overshadows all other symptoms, leading to the complete ignoring of the primary condition. Especially is this true of the pneumonia of alcoholic subjects. In the latter case it is very easy to fall into the error of diagnosing the case as one of delirium tremens. The only way to avoid this pitfall is to make routine physical examinations of the chest in all cases.

The delirium of acute inflammatory rheumatism is now believed to be the result of hyperpyrexia. At one time it was held that it was evidence of recession of the disease to the meninges. Autopsies have failed to establish this relationship, for hyperpyrexia has been observed in all the reliably recorded cases, and the delirium has disappeared on the return of a comparatively low temperature.

The delirium of *sunstroke* is likewise a probable sequence of the hyperpyrexia, for it disappears promptly on the lowering of the bodily temperature by cold baths.

The assumption that the delirium of erysipelas is due to involvement of the meninges is not supported by post-mortem examinations. It is probably the result of the combined action of fever and toxæmia.

Delirium is one of the less commonly recognized phases of uræmia. The diagnosis, of course, depends upon the recognition of renal disease by reason of the state of the patient's urine. At the same time, the observer must take care to determine that all the factors demonstrating renal disease and function sufficiently diminished to produce uræmia are present ; for it is not uncommon to find albuminuria associated with delirium, just as it may attend other cerebral states, *e.g.*, apoplexy. The delirium of uræmia may be mild or wild ; it may resemble the typhoid state ; in fact it might readily be mistaken for the same but for the subnormal temperature and the history of the illness. Sometimes the delirium is so wild as to give rise to the diagnosis of acute mania.

3. **Delirium and Inanition.**—It is well known that one of the symptoms of prolonged starvation is delirium. This symptom, under these circumstances, has been explained on the assumption of cerebral irritation dependent upon anæmia. Some observers claim that there is a "fluxional hyperæmia of the brain." In some of the cases the period of actual delirium has been followed by one of excitement characterized by delusions. The anxiety and suffering accompanying a period of starvation must influence greatly the nervous system, and these factors undoubtedly play some part in the production of symptoms. In some of the acute diseases the delirium of the later stages is to be regarded in part, at least, as due to inanition, and is a strong indication for stimulation and additional nutrition. The delirium occurring in the terminal stages of fatal anæmias is dependent in part upon the deprivation of the brain of blood, and in part upon the resulting changes in that organ from such a condition.

(*d*) **Neuropathic Delirium.**—Under this heading may be considered those deliriums symptomatic of certain nervous affections, and some of the insanities characterized by incoherence of thought, speech and action, and generally known as mania. Of the nervous affections, especial attention should be paid to epilepsy and hysteria. In some few cases of the former disease delirium takes the place of the convulsion. As in all cases of epilepsy., there is absolute unconsciousness, and the attack comes on without apparent cause. Following the mental exaltation, the patient usually goes into a deep sleep, as in ordinary epilepsy. In still other cases the delirium succeeds the convulsion ; indeed, may constitute a veritable mania. Another manifestation of mental disturbance in epileptics is found in interparoxysmal symptoms, consisting for the most part of boisterous or obstreperous conduct, during which the patient may become destructive. These latter cases are undoubtedly the product of the mental degeneration which sooner or later appears in nearly all epileptics, independently of the use of bromides. In some few, however, there seems to be a direct relationship between the mental condition and the exhibition of the medicine. I am inclined to the view, however, that in most of such instances the convulsive seizures act as a sort of safety valve, and that it is the suppression of the paroxysms, and not the medicine *per se*, that is responsible for the mental state. At any rate, it is worthy of note that the suspension of the medicine and the occurrence of an attack seems to have a civilizing effect upon the sufferer.

The delirium of the hysterical is responsible for many ridiculous diagnostic errors. The condition is hard to describe. And yet any one who has seen much of hysterical patients can recognize the nature of the case at a glance. As negative evidence, we have the absence of the symptoms observed in other deliriums and manias which tell the observer that the patient is really sick, and the positive evidence of a history of the neuropathic temperament, surroundings capable of generating hysteria, the manner of onset, the emotional cause, the coördinate movements and ravings, etc. Then, too, we are unable to account for the symptoms present on any other hypothesis than that of hysteria.

Delirium may be symptomatic of any organic brain affection, as meningitis, tumor, abscess, hæmorrhage, etc. In meningitis it is practically constant. Indeed, we regard it as one of the four cardinal symptoms of the disease, the remaining three being fever, headache, and vomiting.

Of the insanities characterized by an exalted mental state we have four of special importance, namely, acute and chronic mania, acute delirium, and general paralysis of the insane.

Acute Mania.—While the main symptoms of an attack of acute mania are of rapid onset, nevertheless careful inquiry always elicits the fact that for several days, sometimes a number of weeks, prior to the outbreak, the patient has given ample evidence that he is not in his normal condition. There may have been noted nothing more than some departures from his usual behavior. This may be so great as to call for comment. It usually, however, consists of some degree of mental depression. Later this gives place to an exhilaration, the patient then being abnormally happy, talkative, and generally optimistic. Finally the symptoms of acute mania appear. Then the patient becomes mentally and physically active. Ideas crowd upon the mind one after another, and without any logical relation. The attendants are made aware of the patient's thoughts by his constant loquacity. The uninitiated, not acquainted with the normal mental state, are commonly bewildered by the clinical picture. Taken singly, many of the patient's sayings are apparently bright and witty; taken in their associations, they become the veriest nonsense. The slightest incident in the sick-room or a casual remark serves as a text for changing the trend of thought. As a rule, the patient has some one thought or series of ideas which stand forth more prominently than others. He may imagine himself to be the Creator, a millionaire, a great benefactor, and considerable of his talk is in keeping with the predominant idea. Hallucinations and illusions are generally absent. Sleep is nearly always absent; indeed, the patient may go for a number of nights without closing his eyes unless a hypnotic is prescribed. One remarkable feature of the language used by the maniacal patients in many instances is its vulgarity. Oaths and obscene words may be expressed in almost every sentence, and this, too, by young women who, in their normal states, would revolt at such ideas.

Along with this excited mental state the patient is usually physically active. He walks about the room more or less constantly. He may even attempt to escape into the street. In some cases he disrobes, regardless of those who may be present. The physical condition varies. With such ceaseless mental and physical activity, and with the comparatively small quantity of food taken, it is not surprising that he emaciates, and that exhaustion ultimately supervenes. The tongue becomes dry, and possibly brown. The bowels are usually constipated.

Cases of acute mania are of variable duration. It is unusual, however, for recovery to take place under three months. Improvement is generally announced by the return of natural sleep and the appearance of lucid intervals, which become more frequent and of longer duration as the patient approaches the normal state. The majority of cases of acute mania recover. Some die by exhaustion; others lapse into a permanent dementia.

Acute Delirium; Bell's Mania.—There is quite a difference of opinion as to whether or not this should be regarded as a disease apart from acute mania. It presents the same mental and physical phenomena as above outlined; but, in addition, there is high fever, the temperature generally ranging around 104° F. The disease usually terminates fatally.

Delirium Tremens.—The patient at the onset of the disorder exhibits symptoms common in chronic alcoholic habitues, as nervousness and restlessness, tremor, insomnia, and dreams of an unpleasant character. In very many instances there is disgust for liquor. This latter symptom may account in great measure for the popular superstition that delirium tremens is the result of a too sudden breaking off of the habit when partaking of large, daily quantities of liquor. The delirium itself usually shows itself first in an undue loquacity. The subjects discussed are handled in a rambling manner. Very often the patient declares himself perfectly well, and demands that he be permitted to go home, or that he may go to his work. Then visual hallucinations appear, and are nearly always of a horrible nature. The patient sees mice, snakes, and other repulsive animals about him. He may even see mobs bent on his destruction. Auditory hallucinations, though less common, are frequently present. The patient's whole manner is in keeping with his false ideas. He exhibits an expression of terror or horror ; he endeavors to escape from his imaginary enemies, even resorting to violence to accomplish his object. This violence is, however, one of defence rather than of offence. While the patient lies in bed he picks restlessly at the bedclothes, his movements being characterized by a very marked tremor. The symptoms are always worse at night ; indeed, they may be present only at such times. The delirium usually continues for five or six days, when it subsides, and the patient slowly regains his strength. In less fortunate cases a condition of exhaustion sets in, the pulse becoming weak and rapid. The most frequent cause of death is heart failure. Fever is commonly present, the temperature rarely rising, however, above 102° to 103° F. It is not unusual to find albuminuria present, even in cases in which there is no organic renal disease. It is more common, however, to find the disease engrafted on a condition already undermined by chronic kidney disease, in which case the urinary condition is greatly aggravated by the delirium tremens.

CHAPTER XIX.

CEREBRAL LOCALIZATION.

THE successful practice of cerebral localization is possible only through a thorough knowledge of the practical anatomy and physiology of the brain, together with an understanding of the underlying principles of pathology. Empirical matching of symptoms in the patient with typical cases outlined in text-books is well-nigh useless, for the different focal lesions distribute themselves through the cerebral substance in kaleidoscopic variety. To succeed, one must possess anatomical, physiological and pathological knowledge, and to these he must add clinical experience. The functions of certain areas are so well understood as to make the recognition of disease limited thereto a not very difficult matter in the majority of instances. Numerous cases, however, will be observed in which lesions of other portions of the brain are impossible of correct diagnosis.

Anatomical Considerations.—For clinical study, the brain may be considered as consisting of surface or cortex, the central white substance or centrum ovale composed of numerous systems of conducting tracts, and the basal ganglia.

The cortex is to be studied as consisting of lobes, lobules, convolutions, fissures, and sulci. These we will study *seriatim*.

Taking first the *lobes*, we find these to be four in number: the frontal, parietal, occipital, and the temporo-sphenoidal.

The fissures are the longitudinal, the Rolandic, the Sylvian, the parieto-occipital, the calloso-marginal, the calcarine, and the parallel.

The Longitudinal Fissure divides the two halves of the cerebrum.

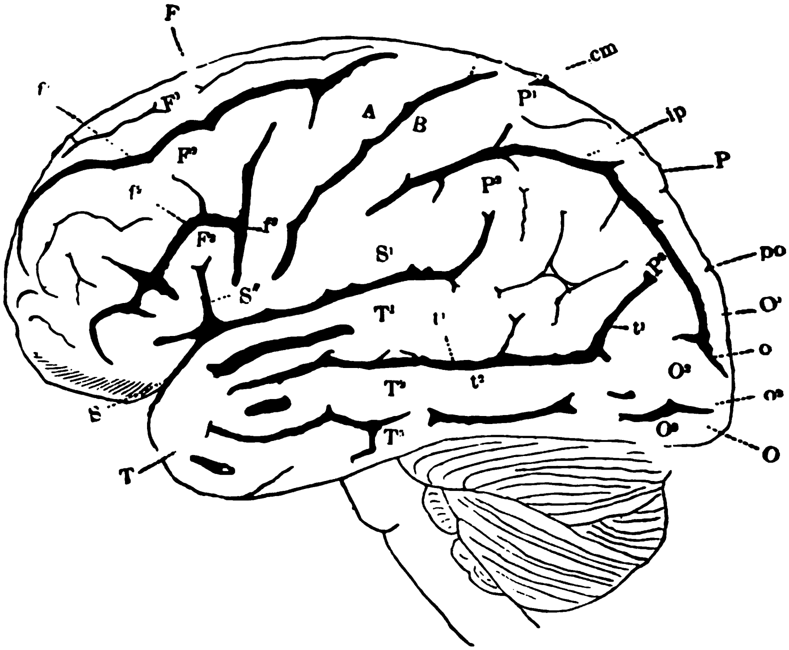
The Fissure of Rolando starts from a point just posterior to the middle of the longitudinal fissure, and extends downwards for a distance of three and three-eighths inches at an angle of sixty-seven degrees. It reaches nearly to, but does not join, the fissure of Sylvius.

The main portion of the *Fissure of Sylvius* runs in a nearly horizontal direction, and separates the temporo-sphenoidal lobe below from the frontal and parietal lobes above. Near its anterior extremity it gives off a small branch, described as the vertical or short limb of the fissure. Between the vertical and horizontal fissures is a portion of cortex known as the operculum.

The Parieto-occipital Fissure is seen mainly on the median surface of the brain, only its outer extremity encroaching on its convexity at a point midway between the upper terminus of the fissure of Rolando and the posterior extremity of the occipital lobe.

The Calcarine Fissure can be seen only on the median surface. It joins the parieto-occipital fissure at its innermost terminus, and extends backwards to the posterior extremity of the brain. The calcarine and parieto-occipital fissures and the outer edge of the median aspect of the brain bound a triangular area called the cuneus.

FIG. 210.



Outer surface of the left hemisphere.—After Ecker.

F, frontal lobe; **P**, parietal lobe; **O**, occipital lobe; **T**, temporo-sphenoidal lobe; **S**, fissure of Sylvius; **S'**, horizontal, **S"**, ascending ramus of the same; **c**, central sulcus or fissure of Rolando; **A**, anterior central, or ascending frontal convolution; **B**, posterior central, or ascending parietal convolution; F^1, superior, F^2, middle, and F^3, inferior frontal convolutions; f^1, superior, f^2, inferior frontal sulcus; f^3, præcentral sulcus; P^1, superior parietal or postero-parietal lobule; P^2, P^3, inferior parietal lobule, viz.: P^2, supra-marginal gyrus; P^3, angular gyrus; **ip**, intraparietal sulcus; **cm**, termination of the calloso-marginal fissure; O^1, first, O^2, second, O^3, third occipital convolutions; **po**, parieto-occipital fissure; o^1, transverse occipital sulcus; o^2, inferior longitudinal occipital sulcus; T^1, first, T^2, second, T^3, third temporo-sphenoidal convolutions; t^1, first, t^2, second temporo-sphenoidal sulci.

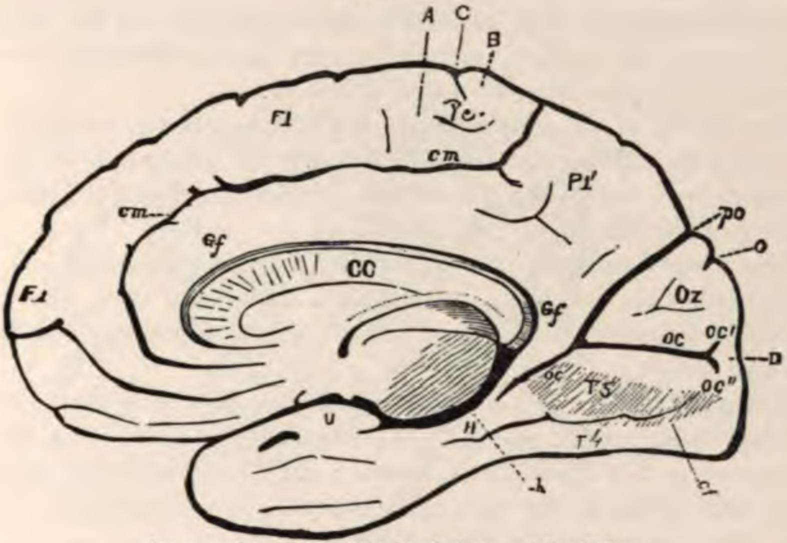
The *Calloso-marginal Fissure* is found on the anterior portion of the median surface of the brain. It lies midway between the upper cerebral margin and the corpus callosum. Its posterior extremity curves upwards.

The *Frontal Lobes* constitute that portion of the cerebrum lying anterior to the fissure of Rolando. The *parietal lobe* includes that portion of the brain lying above the Sylvian fissure and between the occipito-parietal and Rolandic fissures. The *occipital lobe* includes all lying posterior to the fissure last named. The *temporo-sphenoidal* lobe lies below the fissure of Sylvius.

Each of these lobes has its convolutions, named according to their position or to some peculiarity in shape. Thus, in the frontal lobe we find the *first*, *second* and *third frontal convolutions*, running in general direction parallel to the longitudinal fissure, the first in number being the nearest to that landmark. On the under surface of this lobe, that is, the portion resting upon the orbital plates, we find adjoining the longitudinal fissure the *gyrus rectus*, next to a convolution in which the olfactory lobe is set.

On either side of the fissure of Rolando are important convolutions, known generally as the *central convolutions*, the so-called motor area. The anterior one is called the *ascending frontal* or the *pre-Rolandic convolution*, and the posterior, the *ascending parietal* or the *post-Rolandic convolution*. Separating the ascending frontal convolutions from the ones in front of it is the *precentral sulcus*. The remaining important convolutions of the parietal lobes are the *supra-marginal* and the *angular gyri*. The supra-marginal convolution lies just above the posterior extremity of the fissure of Sylvius. The angular gyrus is situated behind the posterior end of the Sylvian fissure, and extends backwards to the occipital lobe and upwards to the intraparietal sulcus. Its ends join the first and second temporal convolutions.

FIG. 211.



Inner surface of the right hemisphere.—After Ecker.

CC, corpus callosum, longitudinally divided; *Gf*, gyrus fornicatus; *H*, gyrus hippocampi; *h*, sulcus hippocampi or dentate fissure; *U*, uncinate gyrus; *cm*, calloso-marginal sulcus; *F*1, median aspect of the first frontal convolution; *c*, terminal portion of the central sulcus, or fissure of Rolando; *A*, ascending frontal, *B*, ascending parietal convolution; *Pc*, paracentral lobule; *P*1', precuneus; *Oz*, cuneus; *po*, parieto-occipital fissure; *o*, transverse occipital sulcus; *oc*, calcarine fissure; *oc'*, superior, *oc''*, inferior ramus of the same; *D*, gyrus descendens; *T*4, gyrus occipito-temporalis lateralis (lobulis fusiformis); *T*5, gyrus occipito-temporalis medialis (lobulus lingualis); *cf*, collateral or occipito-temporal fissure.

The temporo-sphenoidal lobe has two, sometimes three convolutions. These run parallel to the Sylvian fissure. The sulcus dividing the *first* and *second temporal convolutions* is called the *parallel fissure.*

On the median aspect of the cerebrum we have two important convolutions in addition to those already mentioned. Between the calloso-marginal fissure and the upper margin of the median surface is the *marginal convolution.* Between the same fissure and the corpus callosum is the *gyrus fornicatus* or the *convolution of the corpus callosum.* Continuous with the gyrus fornicatus and extending to the temporo-sphenoidal lobe is the *hippocampal convolution.*

The *uncinate* and *lingual gyri* are on the median surface of the temporo-sphenoidal lobe. The uncinate is the upper one, and has been so named because its anterior extremity curves around like a hook. The lingual gyrus lies just below the calcarine fissure.

The *lobules* of the cerebrum are the paracentral, the quadrate, the cuneus, and the superior and inferior parietal. The quadrate is also called the precuneus; it lies between the occipito-parietal fissure and the upcurved extremity of the calloso-marginal fissure.

The *superior parietal lobule* forms that portion of the parietal lobe into which extends the ascending parietal convolution, and lying above the interparietal fissure or sulcus. The *inferior parietal lobule* lies immediately below that fissure.

About the anterior end of the fissure of Sylvius is an area of unusual physiological importance, owing to its relations to the functions of speech. The operculum has already been mentioned. Lying within the fissure of Sylvius and hidden by the operculum is the *island of Reil,* which consists of from five to seven small convolutions.

The *centrum ovale,* the white substance of the brain, consists of conducting tracts. These may be divided into three classes or systems: (1) projection fibres; (2) commissural fibres; and (3) association fibres. The projection fibres or tracts transmit motor and sensory impulses from the cortex to the spinal cord. The commissural fibres connect corresponding points in each lateral half of the cerebrum with each other. The association tracts run between adjacent portions of the cortex.

The *basal ganglia* are the optic thalami, the corpora striata, the corpora quadrigemina, and the corpora geniculata.

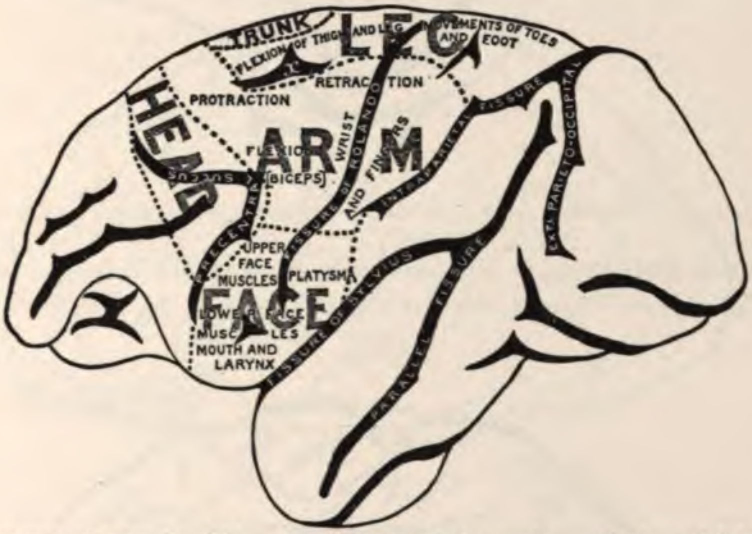
Of these the corpora striata are of the greatest physiological importance. Each of these bodies consists of two masses of gray matter separated by a band of white. The former are known as the caudate and lenticular nuclei, and the band of white between as the internal capsule. This internal capsule is a portion of the projection system of fibres, for it contains the fibres extending from the cortex to the crura. It is divided into two portions, which are united by a sharp bend,—the knee of the capsule.

The anterior portion of the capsule transmits fibres from the frontal lobes only. The posterior limb contains the fibres from the motor area and the occipital lobe.

The *corpora quadrigemina* are masses of gray matter lying immediately above the aqueduct of Sylvius, and beneath the posterior portion of the corpus callosum. The anterior pair are the larger, and are called the nates; the posterior, the testes. They are connected with the optic thalami and the optic tracts.

The *corpora geniculata* are two masses placed "on the outer side of the corpora quadrigemina and on the under and back part of each optic thalamus, and named, from their position, corpus geniculatum externum and internum. They are placed one on the outer and the other on the inner side of each optic tract."

FIG. 212.



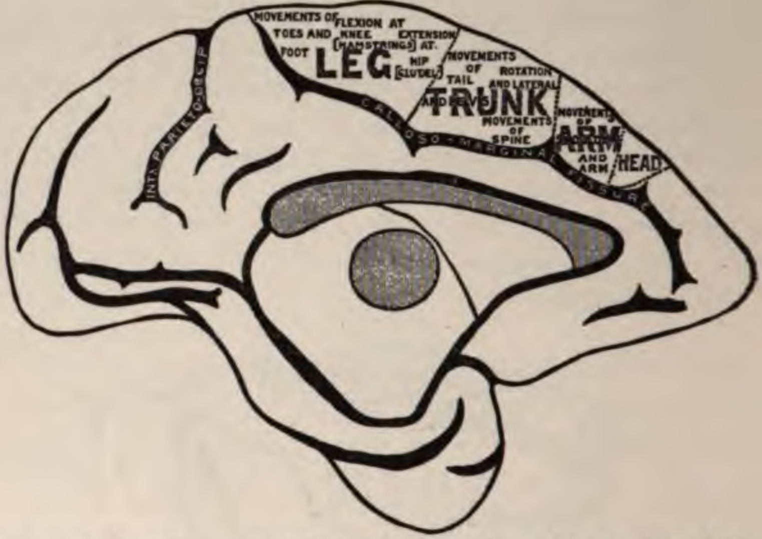
Motor areas of convexity of the cortex of the brain in the monkey.—Horsley and Schäfer.

Physiological Considerations.—The functions of all the parts above mentioned have not, by any means, been sufficiently elucidated. Of quite a number of them it may be said that we know absolutely nothing. There are some situations in which quite large lesions give rise to no symptoms whatever. These are generally referred to as the silent areas of the brain.

For the present, the frontal lobes must be regarded as belonging to these silent areas; for, while we are well aware of the fact that they in some way subserve mental processes, we also know that they may be very extensively diseased without giving rise to any trouble.

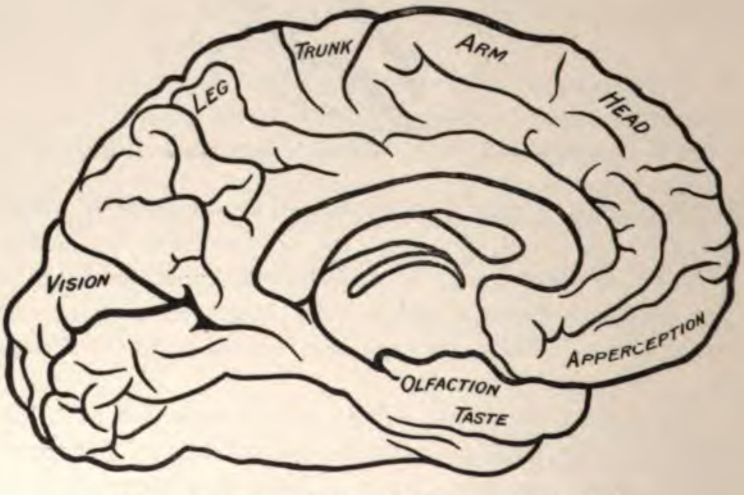
The most interesting, probably because the best understood, is the motor area of the cortex. This, as already stated, is composed of the ascending frontal and ascending parietal convolutions, together with the upper extremity of the first frontal convolution, and the paracentral and superior parietal lobules. The muscles of the legs find their centres in the upper portion of this area, the arm centres being in the middle on either side of the Rolandic fissure, while the face finds central representation in the lowest of all. The expression above used, specifying a representation

FIG. 213.



Motor areas of the median aspect of the brain in the monkey.—After Horsley and Schäfer.

FIG. 214.



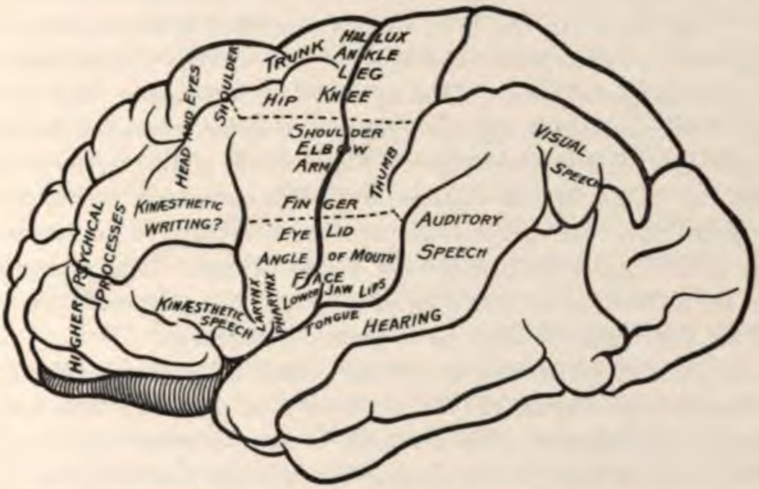
The cortical centres of the median aspect of the brain.

of the muscles of a part, does not exactly specify the correct idea, as it is not muscles but movements that are represented in the cortex.

In assigning to each convolution its appropriate functions a caution is necessary. While the surface of the brain is spoken of as consisting of fissures and convolutions, it must be remembered that these are but relative terms, and that the bottoms of the fissures and sulci will be found to contain the same layers of gray matter as surmount the convolutions. It must also be borne in mind that the sulci and fissures do not act as boundary lines for definite functional parts.

Taking, first, the leg centres, we find them occupying the convolutions bordering on the longitudinal fissure, both on the median and outer surface of the brain. Different authorities assign the centres for each movement somewhat differently. The centres for the hip seem to lie most anteriorly, that is, in the ascending frontal and upper portion of the first frontal convolution. Those for the hip, knee and ankle occupy the upper extremity of the ascending frontal, those for the great toe the upper portion of the ascending parietal, and those for the smaller toes the space between the last-mentioned area and the parieto-occipital fissure.

FIG. 215.



The cortical centres of the convexity of the brain.

The arm centres lie on either side of the fissure of Rolando, in the motor convolutions. Taking them from above downwards, they occupy the middle two-fourths of these in the following order: shoulder, elbow, fingers, index finger, and thumb.

The face centres are found in the very lowest portion of the frontal convolutions, occupying mainly, however, the ascending frontal.

The centres for the trunk muscles, if we are to rely upon the teachings of Horsley and Schäfer, are to be placed upon the inner surface of the hemispheres. This statement is not to be accepted as positively reliable. Much uncertainty must exist as to these centres, for the reason that the functions subserved by these muscles are such as to require bilateral action, and, therefore, bilateral representation in the cortex. Of this, more will be said hereafter.

A centre for the upper eyelid is believed to exist in the angular gyrus.

The centre for the pharynx and the larynx has been assigned to the lowermost portion of the ascending convolution bordering on the fissure of Sylvius.

The situation of the sensory centres has not yet been positively determined. The weight of clinical and experimental evidence favors the view that the motor area is likewise a sensory one.

The most important function subserved by the occipital lobe is that of vision. That portion of this lobe, already described as the cuneus, contains the sight centres for the same-named half of each retina. Thus a lesion of the right cuneus causes blindness of the right half of each retina. Anterior to the occipital lobe, and in the angular gyrus, is a higher visual centre, in which both half-fields find representation. Destruction of this area causes obscuration of vision in the opposite eye, with concentric limitation of the visual field. The eye of the same side seems to have representation also, for the lesion seems to damage the vision of that. The primary visual centres are located in the posterior part of the optic thalamus, the external geniculate body, and the anterior corpora quadrigemina.

The sense of smell finds its cortical centre at the anterior extremity of the uncinate convolution, on the same side as that on which function is abolished. The primary olfactory centre is the olfactory lobe.

The sense of taste has its cortical centre in the lingual gyrus.

The auditory centre is in the hinder part of the first and possibly of the second temporal convolutions. The primary centres are believed to be located in the posterior corpora quadrigemina and the internal geniculate bodies.

Principles Involved in Localization.—As already stated in these pages, the correct understanding of a given case of cerebral disease involves the determination of two important points: the first is the nature of the lesion, and the second its location. A satisfactory conclusion respecting these can only be reached by a careful study and a logical consideration of all the data obtainable. The determination of the former is often of great importance in assisting to that of the latter point.

To illustrate: It is well known that the most frequent cause of cerebral hæmorrhage,—indeed, the usual cause,—in people who have passed middle life, is rupture of miliary aneurisms, and this rupture takes place almost invariably in the lenticulo-striate artery. If, then, we have the symptoms present showing such a pathological condition, and if we have complete hemiplegia, we are justified in diagnosing capsular lesion, although similar symptoms may arise from lesion elsewhere. Still another illustration may be found in connection with disease of the ear. If the symptoms point strongly to the presence of cerebral abscess, it is a matter of experience that that abscess is nearly always either in the temporo-sphenoidal lobe or in the cerebellum. If the mastoid is diseased, the cerebellum is more likely to be the seat of trouble.

All symptoms from focal brain disease are not of equal value for localization purposes. To aid in the consideration of the subject, symptoms may be considered as direct and indirect. The direct symptoms are those resulting from the actual damage done by the lesion within its limited area. The indirect symptoms are those arising by reason of the interference of the functions of distant or neighboring parts. This it may do by the very sudden onset of the lesion, the interference with the cerebral circulation usually occurring under such circumstances, generally exerting a profound and widespread impression; a tumor by its mechanical action compresses or irritates adjacent parts. In the case of hæmorrhage, the brain recovers from the shock, and the indirect symptoms disappear, leaving a symptom or series of symptoms dependent solely upon the local destruction by the clot. These only are of value for localization purposes.

It is important always to study the march of the symptoms. Taking a tumor in the motor area by way of illustration, let us presume that the convulsion first involves the thumb only. Later the spasms are more widespread. In subsequent attacks the movement covers increasingly larger areas because of the natural increase of the growth. Then come headache and general symptoms. The important symptom is the spasm of the thumb. It shows the starting-point of the lesion. The initial localizing symptom has been very appropriately called the signal symptom.

Lesions must be regarded as producing two classes of direct symptoms, the destructive and the irritative. In the clinical investigation of each case, it is necessary to assign every symptom to either of these classes.

It is important to bear in mind that muscles accustomed to act bilaterally have bilateral representation in the cerebral cortex. The muscles of the trunk, as also those of the larynx and pharynx, are of this order. In case of a destructive lesion affecting the centres for these parts, but little if any noticeable loss of power results. The reason for this is found in the fact that the healthy hemisphere at once assumes the function of the injured one. Compensation takes place at once. Certain muscles have but partial bilateral representation; for example, those of the hip and shoulder-joints. The legs have better bilateral representation than the arms; a condition one is led to expect, because the legs are accustomed to act in association, as in walking. Highly specialized movements, as those of the fingers, have representation on one side only. When they are paralyzed, compensation is not likely to take place.

In convulsions dependent upon lesion of one hemisphere, muscles having bilateral representation are likely to be bilaterally affected.

Lesions of the Frontal Lobes.—It has already been stated that the probable function of the frontal lobes is mental. In unilateral lesions but little psychical disturbance follows, as a rule, probably because the healthy side compensates for the loss on the other. When actual mental impairment is not prominent there may be noticed some changes in the patient's character, as peevishness, inability to concentrate thought, inattention, etc. A lesion at the posterior part of the upper frontal convolution causes symptoms referred to the leg of the opposite side of the body. A centre for the movement of the head and eyes has been described as situated at the base of the second frontal convolution. A lesion here rarely produces permanent impairment of these movements, for the opposite side of the brain compensates rapidly for the loss. Some unsteadiness in walking, similar to that observed in cerebellar trouble, has been observed as occurring sometimes in frontal lobe disease.

Quite recently Phelps has reported a series of cases tending to prove that interference with the functions of the left frontal lobe alone produces mental impairment.

Lesions of the Motor Area.—According as the lesions are destructive or irritative, the symptoms are paralytic or convulsive. The distribution of these will vary according to the limitation of the lesion. Lesions producing localized convulsions are nearly always cortical. The cortical origin of such lesions becomes a certainty when the paroxysms are observed to recur from time to time, always presenting the same type. A convulsion from cortical lesion may spread and involve all the parts tributary to that side. In paralyzing lesions, on the other hand, the loss of power is in strict accordance with the destroying lesion.

Lesions of the Temporo-Sphenoidal Lobe.—Aside from the fact that auditory functions originate in the temporo-sphenoidal lobes, our knowledge of this portion of the brain is as yet uncertain. Destruction of the posterior third of the first and possibly of the second temporal convolution produces deafness of the opposite and impaired hearing of the corresponding ear. This auditory defect soon disappears, however, because each auditory centre is connected with both acoustic nerves, and the healthy side soon compensates for the one that has been injured. Chronic lesions in this locality, therefore, produce no auditory symptoms. The deafness resulting from acute lesions, *e.g.*, hæmorrhage, follows the course above outlined, *i.e.*, early recovery of hearing by reason of compensation on the part of the normal side.

The above remarks apply to destructive lesions in this locality. Irritation causes auditory hallucinations, and even convulsions preceded by auditory auræ. It may also excite conjugate deviation of the head and eyes, but such movement is probably dependent upon auditory impressions.

It seems to be settled with a fair degree of certainty that the olfactory and gustatory functions have their centres in the temporo-sphenoidal lobes. Their exact location, however, cannot be said to have been positively settled. Zuckerkandl contends that the entire limbic lobe takes part in olfaction. Others teach that it is limited to the posterior part of the hippocampal convolution.

The intimate relation between the senses of taste and smell in man suggests that their centres are closely associated in the brain. The limited facts at our disposal indicate that this suggestion is true, and that the gustatory centre is located in the cortex of the inferior temporal convolution on its internal surface, and below the hippocampal gyrus and the gyrus uncinatus.

Lesions of the Zone of Language.—The special clinical feature of a lesion in the zone of language is aphasia. The study of this subject is beset with difficulties, which seem to have arisen from the numerous attempts made to manufacture plausible theories concerning the why and wherefore of this interesting symptom. Authors, in handling the subject, delve too much in psychological discussion, thereby so beclouding the practical facts as to make the general practitioner abandon a study of the problem in a brain-weary condition. Whether the writer will succeed in presenting a clear exposition of the subject is another matter; he will certainly do his best,—acknowledging, however, that there is not a subject which has been treated in this work that has caused him more misgivings in the presentation thereof.

Starting with basic facts, we first state the definition of aphasia as follows:

Aphasia includes all defects characterized by an inability to externalize ideas in words or language, symbols, written character or gestures; and, though vision and hearing be perfect, inability to appreciate ideas from words conveyed to him by these senses. This constitutes a complete aphasia.

If the patient is unable to externalize ideas in speech, symbols, or gestures, the defect is emissive. We call it, for sake of convenience, "**motor aphasia.**"

If the defect is on the receptive side, *i.e.*, if the patient is unable to appreciate or understand printed or spoken words, we call it **sensory aphasia.** **Apraxia** is a term used as synonymous with sensory aphasia.

If the patient is unable to convey ideas by writing, we have the condition known as **agraphia.**

If the patient is unable to understand spoken speech although hearing is intact, the condition present is "**word-deafness.**"

If he is unable to understand written speech, vision being normal, the condition is called "**word-blindness.**" "**Alexia**" is synonymous with word-deafness.

Dyslexia is a term used with such a variety of meanings as to make it a medium of confusion rather than of enlightenment. Gowers describes it as a paroxysmally recurring difficulty in reading. Berlin defines it as a morbid dread of reading. Both authorities regard it as a serious symptom, believing it to be evidence of degenerative cerebral changes. Billings makes both it and alexia synonymous with word-blindness.

An inability to convey ideas by gesture is known as **amimia.**

Physiologically, no speech processes are motor, although we have one variety of aphasia which we designate by that title. The various centres are memory storehouses. They are three in number, including motor, visual, and auditory speech memories. The conducting fibres proceeding from these do not belong to any projection system of the motor tract. The so-called motor speech area, which lies at the posterior part of the base of the third left frontal convolution, by reason of the articulatory memories stored therein, when occasion demands it sends impulses to the Rolandic area. Through these impulses the various movements involving respiration, vocalization, and action of the tongue and lips, are coördinated for the performance of articulate speech.

The speech-centres in all varieties of aphasia are situated in the left half of the brain in right-handed individuals, and in the right half in the left-handed. Exceptions have been reported apparently proving the incorrectness of this assertion, based upon the fact that the patient used his right hand in writing, and in the performance of other acts. But in all such instances further inquiry has developed the fact that, after all, the patient was left-handed, and that the frequent use of the right hand was the result of training.

The right half of the brain is capable of education as a speech-centre, but the possibility of such training is limited, excepting in children. When compensation takes place in this way, a second lesion occurring in the speech-centres on the right side, the aphasia becomes permanent.

The general shape of the zone of language has been compared to that of a gondola, and is well illustrated in Fig. 216. Within this area we find three speech-centres, namely, the centre for articulatory memories, that for visual memories, and that for auditory memories. The position of the centre for articulatory memories has already been described. Lesions of it produce so-called motor aphasia.

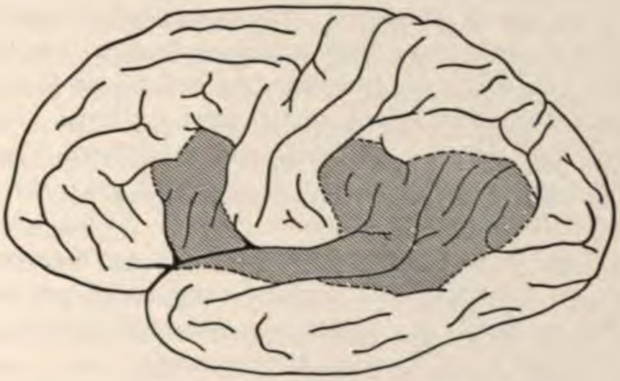
The centre for visual memories is situated in the angular gyrus. Lesions of it produce word-blindness or visual aphasia.

The centre for auditory memories is located in the first temporal convolution about the temporo-parietal sulcus. Lesions of it give rise to word-deafness or auditory aphasia.

It is next in order to describe the clinical features of the several varieties of aphasia, and the methods best adapted for their diagnosis.

1. **Motor Aphasia.**—Two forms of motor aphasia have been described, namely, the cortical and the subcortical. They are to be differentiated, clinically, only after a painstaking investigation. In both forms, the ability to communicate thought by language is lost. Cortical motor aphasia exhibits an inability to speak spontaneously or to repeat words after another. The ability to comprehend spoken speech is preserved. The muscles governing the movements of articulations have their functions unimpaired. The ability to express thought in other ways than by articulate speech is also lost, so that the patient is unable to read aloud, to write, and to convey ideas by gestures.

FIG. 216.



The zone of language.—After Mills.

It may happen that the aphasia is not complete, *i.e.*, the patient retains the ability to utter certain words. When such is the case, the latter are monosyllabic, or they may be expressive of emotion, or, as is sometimes the case, the last words uttered by the patient at the time of the onset of the attack. Thus the patient may be able to say "Yes" and "No"; or he may utter his favorite oath.

Sometimes the aphasia is very limited, the patient having lost the ability to utter substantives.

Agraphia, as already stated, is almost invariably associated with motor aphasia. It may happen, however, that he has preserved the ability to write his name and address, and some few other words, which he has written so frequently in the past as to make them mere automatic utterances. The patient can usually write from copy.

Subcortical Motor Aphasia.—This presents the same clinical features as cortical motor aphasia, with the exception that the patient is able to write in proportion to the preservation of speech, and he can respond to the Proust-Lichtheim test. In subcortical motor aphasia the patient has in his mind the name of the object which he is incapable of emitting. To demonstrate this, Proust and Lichtheim suggested that the patient be asked to name the number of syllables in a word he had heard, and to indicate his answer by compressing the examiner's hand as many times as there were syllables in the word.

2. **Auditory Aphasia; Word-Deafness.**—The centre for auditory word memories lies, as already stated, in the cortex of the middle and posterior portions of the first temporal convolution, extending upward into the supramarginal gyrus. It thus lies between the areas for articulatory and visual word memories, and is rarely involved without evidence of disturbance in the other speech processes. The symptoms of auditory aphasia include inability to form ideas from words spoken to him, although he hears the voice distinctly. In some cases the defect is so profound that he fails to recognize the nature of any sound (*mind-deafness*). The patient is unable to write from dictation; he does not comprehend what he reads, etc. In some cases the defect may be so mild that great care is required to determine the nature of the condition present. When conducting the examination, it should be the aim of the physician to discover the ability of the patient to understand the spoken name of objects seen, heard, handled, tasted or smelt; the power to understand speech and musical tone; and the power to call to mind objects named.

3. **Visual Aphasia.**—The symptoms of visual aphasia include an inability to understand the significance of printed or written words, notwithstanding the normal condition of the organs of vision. In cases in which the lesion is not complete, the patient may retain the power to understand letters, but not words. It may also happen that certain words which he sees so frequently that their recognition has become automatic, *e.g.*, his own name, especially when written, are perfectly understood. In case of complete destruction of the angular gyrus, the power of writing is lost. If visual aphasia is uncomplicated, he can understand spoken words. If a sharp instrument is employed to write letters and words on his hands, he can understand them.

As a sub-variety of visual aphasia we have mind-blindness, in which the patient is unable to understand the nature or uses of any object seen. In another variety, known as optic aphasia, he cannot call up the name of objects seen; in other words, he cannot name things.

Subcortical Sensory Aphasia.—Both auditory and visual aphasia may result from subcortical lesions. In **subcortical auditory aphasia** the patient cannot understand spoken words, and he cannot write to dictation; but he can speak spontaneously, can read aloud, can write voluntarily, and can copy.

Subcortical visual aphasia is characterized by word-blindness associated with right lateral homonymous hemianopsia; but the patient can speak spontaneously, can write voluntarily and from dictation, and can understand what is said to him.

The Clinical Relations of Motor and Sensory Aphasia.—Owing to the intimate anatomical relations existing between the centre for articulatory memories and the Rolandic region, motor aphasia is almost invariably associated with right-sided hemiplegia. The zone of language is supplied by the middle meningeal artery, a lesion of which renders it entirely functionless. Hence complete aphasia may be produced. Nevertheless, the lesion may be limited, in which case motor aphasia exists apart from involvement of the other speech areas. As a rule, in fact it might be said almost invariably, motor aphasia is of rapid onset, and is associated with the ordinary symptoms of apoplectic seizures, etc., the aphasia not being recognized until the patient rallies. Sensory aphasia, on the other hand, is generally the result of focal lesions other than circulatory, including tumors and degenerations. The symptoms are of gradual onset, and the speech disturbance occurs independently of involvement of other portions of the zone of language.

Lesions of the Centrum Ovale.—Lesions of the centrum ovale are both rare and difficult of recognition. This portion of the brain is composed of both projection and commissural systems of fibres. Destructive lesions of the former give rise to distinct symptoms, which will resemble those of cortical or capsular origin according as they approach the cerebral cortex or the internal capsule. Lesions involving commissural fibres may give rise to no symptoms, *i.e.*, to no symptoms which we are likely to recognize.

Lesions of the Motor Fibres of the Centrum Ovale.—These produce paralysis of great or small distribution on the side of the body opposite to that of the lesion. If the latter be situated adjacent to the cortex where the different fibres are well separated the paralysis will be limited, as in cortical lesions. When, on the other hand, the lesion is deeply situated and near the internal capsule, a small lesion is capable of producing a widespread hemiplegia. In subcortical lesions there may be spasm of the paralyzed extremities, tonic predominating over clonic, with but moderate or late-appearing headache. In cortical lesion headache is severe, appears early, and is associated with local tenderness, and the convulsions are epileptiform in type, and are succeeded by paralysis in the previously convulsed parts.

Lesions of the Sensory Fibres of the Centrum Ovale.—Mills describes three sets of system fibres of sensory functions as follows: "(1) The bundle which conveys the impulses concerned in common sensibility, the fibres of this bundle probably reaching or approaching the cortex in the limbic lobe and postero-parietal region; (2) gustatory fibres which pass to the lower and more inferior portions of the temporal lobe, probably chiefly to the fourth temporal convolution; (3) a bundle or set of fibres which, proceeding from the lateral fillet, turns downwards so as to take its course into the superior temporal convolutions, conveying to them auditory excitations." Lesions of these several tracts give rise to anæsthesia, word-deafness and other interference with hearing, and disorders of the sense of taste respectively. Mention may be made at this place of the optic radiations of Gratiolet, which continue the projection system of the visual sense and extend from the cuneus to the internal capsule, and disease of which produces hemianopsia, which is more complete as the lesion recedes from the cortical centres of vision. For further data bearing on disease in the latter locality the reader is referred to the section on Hemianopsia.

As to the lesions of the association tracts, practically little is known of clinical value. Most subcortical lesions must produce some interference with commissural and association fibres, and it is reasonable to believe that symptoms must be produced thereby. It remains for future investigators to determine what these symptoms are, and the special structures, lesions of which produce them. Of late considerable attention has been paid to the subject of a stereognosis, *i.e.*, the power of recognizing the nature and use of objects by the sense of touch. This faculty can hardly be dependent upon mere sensation, but rather upon the coördination of various senses,—that is, those of posture, space, pressure, temperature and position. As to which of these sensations is the most important in any given case will depend very largely upon the nature of the object. Thus, the temperature sense will be of paramount importance in recognizing ice; pressure sense in recognizing objects one of the characteristics of which is high or low specific gravity, and so on. Of course, if common sensation is completely lost, the patient will not be able to recognize objects by the sense of touch; but in such cases the trouble is tactile anæsthesia, and not astereognosis, and may result from lesion in almost any part of the sensory pathways. But given a case in which there is comparatively little tactile anæsthesia, and there is inability to recognize the nature of an object by touch, we may safely diagnose the presence of astereognosis. In such cases, the lesion is subcortical and in the parietal region. Already two cases of tumor in this situation have been correctly localized on this symptom.

Lesions of the Corpora Quadrigemina.—Notwithstanding positive statements of authors to the contrary, lesions of the corpora quadrigemina are not easy of recognition, because it is in exceptional instances only that these structures are alone involved. When we bear in mind that the chief lesion in this locality is tumor, which may produce symptoms referred to the corpora quadrigemina, and others by pressure upon the surrounding structures, one can readily see the diagnostic difficulties. The anterior tubercles of the corpora quadrigemina have to do with the reflex movements of the pupil and ciliary muscle. The posterior tubercles and the internal geniculate body have some connection with the auditory nerve, and apparently have some fibres of coördination tracts (some say cerebellar tracts) connected with them. The nuclei of the third nerves lie immediately beneath the bodies.

Lesions of the corpora quadrigemina produce, as an initial symptom, incoördination of movement, the ataxia simulating very closely that arising from cerebellar disease. Next there appears paralysis of the eye-muscles, the superior and inferior recti bearing the brunt. The ataxia is aggravated on voluntary movement. Symptoms referable to the upper extremities include ataxia of movement with intention tremor.

In case of disease of the anterior bodies, pupillary action is lost. This symptom is not always reliable, however, because tumors in this locality are commonly associated with optic neuritis, which is capable of producing blindness and pupillary inactivity. Nevertheless, it is commonly admitted that more or less disturbance of vision, apart from ocular lesion, commonly exists. There will also be the associated ataxia and oculo-motor palsy.

In disease of the posterior quadrigeminal bodies, the pupils react normally, and vision is but slightly, if at all, disturbed. Disturbance of hearing with incoördination of movement and oculo-motor paralysis make up the symptom complex.

Lesions of the Corpora Striata.—Each of these ganglia consists of three parts,—the caudate and lenticular nuclei and the internal capsule. Disease of the caudate and lenticular nuclei is, so far as is known, incapable of producing disturbance of function of clinical importance. An exception to this statement may be taken in those cases in which the integrity of the internal capsule is directly or indirectly affected by the lesion. It is known that loss of motion and sensation may occur in conjunction with lesions in this location, but these symptoms are but temporary, and even then are believed to be due to disturbance of the internal capsule. Experimental observations of Hale White seem to show that in rabbits, at least, the striate bodies have the power of modifying the temperature of the body. Clinically, numerous cases have been observed in which remarkable rises of temperature have apparently been dependent upon disease in this location. In some cases of lesion of the lenticular nucleus, anarthria (pseudobulbar paralysis) has been observed. The occasional association of mobile spasm with lesions of the posterior portion of the lenticular nucleus has been suggested with good reason.

The internal capsule is, on the other hand, a most important structure from a clinical standpoint. In it are concentrated within a small space most of the motor and sensory fibres, as well as those of other important fasciculi. It is generally described as consisting of an anterior and posterior limb united at an obtuse angle, known as the knee of the capsule. The clinical evidence of disease of the anterior limb is as yet wanting. It is known that it consists anteriorly of a bundle of fibres to the thalamus, and posterior to this, and adjoining the knee of the capsule, the frontopontine tract. Lesions in this situation are very rare. In the knee of the capsule are the fasciculi for the motor cranial nerves. Then in the posterior limb we have first the fibres of the pyramidal tract, lesion of which produces paralysis of the lower part of the face and the extremities on the opposite side of the body. Defect of speech occurs at first, but soon disappears. If the lesion is on the left side, there may be temporary aphasia. The paralysis is permanent, and is practically always followed by some degree of rigidity of the affected limbs. The hemiplegia is not necessarily complete, though usually so, for the lesion may involve the fibres going to but one limb. A lesion of the posterior third of the posterior limb produces hemianæsthesia, and in some cases hemianopsia also. A partial lesion affecting the sensory fibres may excite widespread pains instead of anæsthesia.

Lesions of the Corpus Callosum.—Anatomical and physiological investigations have not yet defined the course of the fibres in the corpus callosum, nor have they determined their functional relations. Hence the clinical study of lesions in it has thus far proven very unsatisfactory. We do know, however, that lesions in the corpus callosum must produce different clinical pictures according to the particular fibres involved. In the case of destructive lesions, we should expect bilateral paralysis or incoordination according to the segment of the callosum involved. Irritative lesions should produce bilateral convulsions. Other symptoms observed in connection with disease in this location include stammering, slowness of speech, change in temperament, strabismus, dilatation of the pupils, and rotation of the head. Bristowe has formulated the following proposition respecting the progress of callosal tumors: "*First*, the occurrence of headache and other vague symptoms of progressive cerebral disease; *second*, the gradual onset of more or less well-marked hemiplegia; *third*, the appearance in a greater or less degree of similar symptoms on the opposite side of the body; *fourth*, the coming on of dementia with drowsiness, loss of speech, difficulty in swallowing, and want of control over the rectum and bladder." Inasmuch as the mental deterioration referred to by Bristowe has been observed only in cases of tumor, and not in softening limited to the callosum, it is fair to presume that it may be dependent upon the pressure exerted by the tumor upon other structures.

Lesions of the Pons Varolii.—The many systems of conducting fibres and the numerous cranial nerve nuclei and roots in the pons and adjacent structures make the symptomatology of lesions in this locality a most varied one.

Taking first the types of paralysis which may result from pontine disease, we find first that a lesion, generally of the upper portion of the pons, may be so situated as to produce a hemiplegia which may be indistinguishable from that arising from lesion of the corresponding internal capsule, excepting that there is loss of the power of conjugate deviation of the eyes towards the side of the lesion.

In a second type of paralysis, dependent upon lesion involving the pyramidal tract above the point at which the fibres to make up the facial nerve decussate, there will be hemiplegia involving the face, arm and leg, on the opposite side of the body.

In still a third variety, for the production of which the lesion must be in the lower third of the pons, *i.e.*, below the level at which the facial fibres decussate, there will be paralysis of the arm and leg on the opposite side of the body, and of the face on the same side as the lesion,—in other words, an alternate hemiplegia.

In each of these three hemiplegic types the symptoms are motor. If, however, the lesion be deeply situated and involve the tegmental fibres of the pons, there will be disturbance of sensation (hemianæsthesia), and in all probability involvement of the nuclei of the sixth nerve as well, producing paralysis of the external rectus.

Again, the lesion may invade the sensory root-fibres of the fifth cranial nerve. Then there will be anæsthesia of the face on the corresponding side, and hemiplegia on the opposite side of the body. In these cases there may sometimes be observed rigidity, spasm, or choreic movements of the extremities.

Disturbance of speech sometimes takes place. This is explained by the fact that the motor speech-paths lie dorsad in the portion of the pyramidal tracts, their destruction giving rise to a condition simulating motor aphasia. Difficulty in articulation and deglutition may be produced for the same reason.

If the middle cerebellar peduncle be involved, there will be vertigo, vomiting and tinnitus, with deafness on the side corresponding to the lesion.

Irritative phenomena are liable to occur in acute lesions, as hæmorrhage. They include convulsions, which are peculiar in that they involve both legs or both arms; sometimes they produce semi-convulsive paroxysms of coughing (Gowers). When the irritation involves the area of the fifth nerve, there may be trismus.

Sometimes hæmorrhage into the pons will cause marked pupillary contraction.

Temperature disturbances are not uncommon in acute pontine lesions.

If the patient survives sufficiently long, the temperature may reach 109° F.

Dorsad of the pons lies the *preoblongata*. In this region is situated the nuclei of the fourth to the eighth cranial nerves, inclusive, together with important conducting tracts. The structures are so complicated, however, and the functions of some are so little known, that a study of lesions in this part is clinically unprofitable.

Lesions of the Crura Cerebri.—The characteristic symptom complex of lesion of one crus cerebri is hemiplegia involving the lower part of the face, the arm and the leg on the opposite side of the body, and the muscles supplied by the third cranial nerve on the same side. This is generally known as alternate hemiplegia of the oculo-motor type, or "Weber's syndrome."

An acquaintance with the anatomy of the parts shows that numerous cases may occur without the symptoms just mentioned. The crura are made up of numerous sensory and motor tracts. The under surface, sometimes referred to as the "*crusta*," is made up mainly of motor fibres; the overlying fibres in the interior are sensory in function, and are known as the "*tegmentum*." Furthermore, dividing the crura into quarters from side to side, we are able to assign different courses and connections for the fibres in each of these divisions. Thus proceeding from without inward, the various groups include fibres from the occipital and temporal lobes to the pons, the pyramidal tract, fibres from the striate body and the posterior part of the frontal convolutions and the anterior central convolution, and, lastly, fibres from the insular region and base of the lenticular nucleus to the upper part of the pons. Bearing these anatomical details in mind, one can readily perceive that the symptoms of crural lesions must vary with the situation and the size of the lesion. The association of hemianæsthesia with the "Weber syndrome" indicates positively that the lesion must be intracrural. If there is no sensory disturbance, it is probably subcrural. If the lesion is subcrural and sufficiently large to reach across the interpeduncular space, then the third nerve on the opposite side may be involved, and the symptom complex includes hemiplegia in association with bilateral motor oculi palsy. According to Gowers, "a very superficial lesion may cause no hemiplegia, but partial palsy of one-third nerve from damage to the root-fibres, with persistent giddiness, supposed to be due to interference with the fibres from the cerebellum."

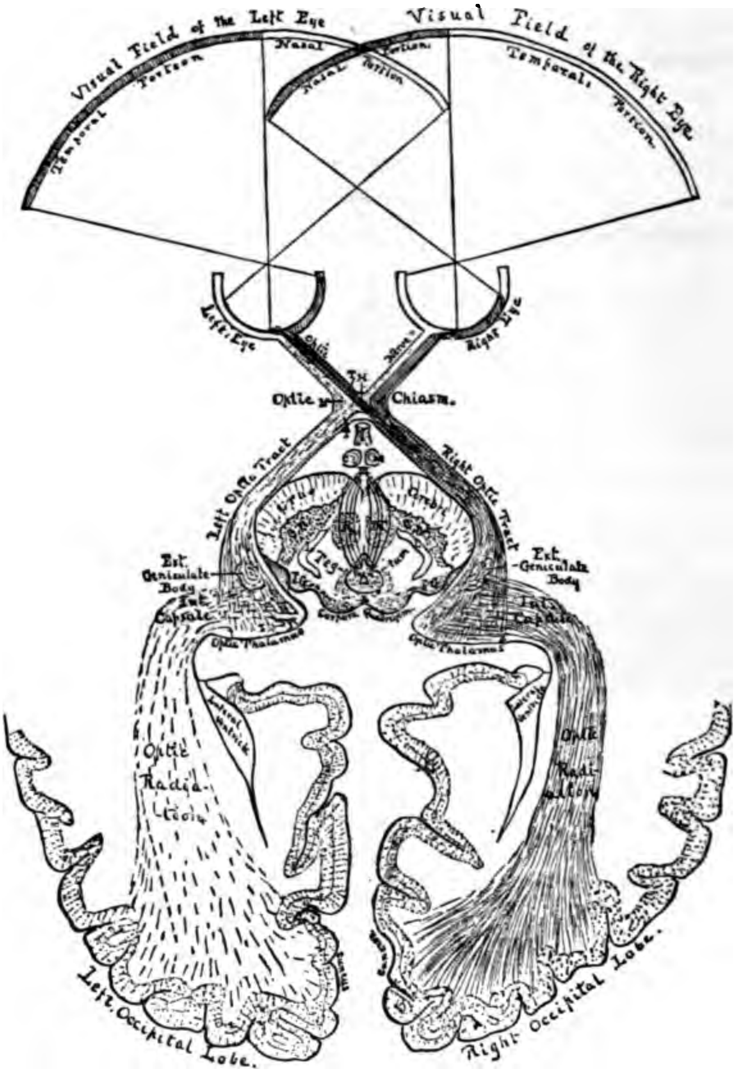
Lesions of the Cerebellum.—Disease or injury of either lateral lobe of the cerebellum produces no symptoms. Lesion of the middle lobe produces incoördination of gait, simulating that of intoxication. The knee-jerks may be either absent or present. Sometimes they are found absent at one stage of the disease, and either present or exaggerated at another. Tumor of the cerebellum, and this is the most frequently encountered lesion in this locality, is apt to be associated with optic neuritis, vomiting and deafness. In many cases of optic neuritis vision is either not at all or but slightly impaired. But in cerebellar disease, loss of vision seems to be more frequently observed. This probably finds an explanation by the pressure exerted on the anterior corpora quadrigemina. The deafness, when present, arises from pressure on the posterior corpora quadrigemina.

Lesions of the Optic Chiasm, Optic Tract and Visual Centres.—The characteristic symptom of a lesion involving the optic commissure or tracts and the cortical visual centres is hemianopsia ; and, conversely, the existence of hemianopsia indicates beyond the possibility of cavil a lesion involving some portion of the structures above named. According, however, as the lesion involves the visual fibres in this or that portion of their course, so will the characteristics of the hemianopsia or its associated conditions vary. So well understood is this fact, that we find in the clinical investigation of hemianopsia a most beautiful and scientific application of the principles of cerebral localization.

By hemianopsia is meant a blindness in one-half of the visual field of one or both eyes. If it affects a half-field in one eye, it is *unilateral ;* if in both eyes, *bilateral.* If the blind half-field is separated from the normal one by a vertical dividing-line, we call it *lateral* or *vertical hemianopsia ;* if the dividing-line is horizontal, *horizontal hemianopsia.* If the blind half-field lies towards the temporal side, it is *temporal hemianopsia ;* if both temporal half-fields are involved, *bi-temporal hemianopsia ;* if the nasal half-field is destroyed, *nasal hemianopsia ;* if the nasal half-field of one eye and the temporal half-field of the other, *homonymous hemianopsia.* The latter term is further qualified by the terms "right" and "left," according to the blind side in its relation to the patient. A unilateral lesion posterior to the chiasm always produces blindness of the same-named half of each retina. It should be remembered, inasmuch as the rays of light cross on entering the eye, that a temporal hemianopsia is due to a blindness of the nasal half of the retina.

A proper practical understanding of the significance of hemianopsia as a symptom requires a knowledge of the course of the visual tracts in the brain. The accompanying diagram, after Starr, adds to the clearness of a written description. On reaching the optic chiasm, each optic nerve divides into two portions. Those fibres which had come from the temporal half of the retina pass on to the tract of the corresponding side ; while those from the nasal half of the retina decussate, and enter the optic tract on the opposite side. "From the optic chiasm each optic tract passes around the crus cerebri and terminates in the external geniculate body, in the optic thalamus, and in the anterior corpus quadrigeminum, as seen in the diagram. From these three masses of gray matter new fibres start out which issue from the outer side of the optic thalamus, enter the internal capsule, and curve backward through the occipital lobe, lying on the outer side of the posterior horn of the lateral ventricle, and thus reach the occipital convolutions, ending chiefly in the cuneus." (Starr.)

FIG. 217.



The optic and visual tracts.—After Starr.

N, Lesion causing nasal hemianopsia; *T*, Lesion causing temporal hemianopsia; *H*, Lesion causing bi-temporal hemianopsia; *C*, Lesion of the optic tract causing left-lateral hemianopsia.

Hemianopsia is not always recognized by the patient. The latter may be fully aware that his sight is not what it should be, but of the exact nature of the defect he may remain in ignorance. In the case of Patrick H., a soldier shot in the occipital lobes at the battle of Antietam, a case that has become historical, the actual state of vision was not known until several years afterwards. The best method of investigating the condition of the visual field,—indeed, the only way for keeping records of a case and comparing the progress made from time to time,—is that with the perimeter. This instrument is not, however, likely to be at the command of the practitioner, and a readier way is necessary. The patient should be seated opposite the physician. One eye should be covered, while the one to be tested should be fixed on a given point of the physician's features,—his nose, or the pupil of one eye. Whatever point be selected for fixation, the greatest care must be observed to have that point immediately opposite the eye to be tested. If the physician's eyes are utilized, it should be the one opposite to that of the patient. The physician must likewise close his eye not in use, that he may judge his own field of vision correctly. Then the physician, holding one hand at a considerable distance to one side, and at the same distance from himself and his patient, slowly brings it inwards until it is seen by the latter. He then tests the power of vision in the same way by approaching the hand from another direction, and then from still another. By comparing the patient's statements with the observations of his own eye, he is able to determine whether there is or is not limitation of the visual field. Finding a limitation, he may make a diagram of the same on a blackboard. The patient is directed to fix his vision on a given spot, while the chalk is approached from the periphery until seen, when that point is marked on the board. The chalk is then approximated from still other directions, and the results noted. By connecting the various points thus obtained, the shape of the field of vision is roughly delineated. During all these investigations, care must be exercised that the patient maintains his gaze on the fixation-point.

I have said that the recognition of the seat of lesion in a given case of hemianopsia is a most interesting clinical problem. We may divide lesions into those in front of and those behind the primary optic centres (external geniculate body, pulvinar of the optic thalamus, and the anterior corpora quadrigemina). Cases arising from lesions in front of these parts, *i.e.*, between these ganglia and the chiasm, present the phenomenon known as the hemianopic pupillary inaction. Lesions behind these ganglia do not affect the pupillary reactions. When the hemianopic pupillary inaction is present, light thrown on the blind half of the retina fails to elicit a pupillary contraction. In applying this test, the greatest care is necessary that the light be thrown on the blind part only; otherwise the observation is valueless. The examination should take place in a darkened room, the source of light should be a small flame, and the ophthalmoscopic mirror should be most carefully handled. The distance at which it is held should be such as to bring the light to the smallest possible point.

When the lesion involves the central portion of the chiasm (Fig. 217, *H*), the decussating fibres are attacked, and bitemporal hemianopsia results.

Should the lesion increase in size and affect the direct fibres on one side, there will be complete blindness on that side, and temporal hemianopsia on the other.

A lesion attacking the outer fibres of the chiasm (Fig. 217, *N*) produces nasal hemianopsia. Symmetrically placed lesions of this character produce binasal hemianopsia, a *very* rarely observed condition. In one reported case this resulted from atheroma of the circle of Willis.

A lesion of one optic tract causes lateral hemianopsia, the side of the hemianopsia being the side opposite to the lesion. It is possible, however, for a lesion affecting one optic tract to involve only a portion of its fibres. Thus a tumor pressing only upon its outer fibres would cause a nasal hemianopsia in the eye of the corresponding side. According to the direction and force with which the pressure is exerted, very irregular types of hemianopsia may result.

In many cases of hemianopsia arising from disease of the tracts or commissure there are associated symptoms indicating involvement of certain cranial nerves or the motor paths, and, not infrequently, changes in the ophthalmoscopic appearances.

If the tract is involved at its termination in the ganglia, the hemianopsia is associated with hemianæsthesia; if in the internal capsule, there will probably be hemianæsthesia and hemiplegia; a lesion between the internal capsule and the cortex will cause hemianopsia with or without hemianæsthesia. A lesion of the cortical centres (in the cuneus) gives rise to hemianopsia alone. The absence of associated symptoms here becomes of localizing value.

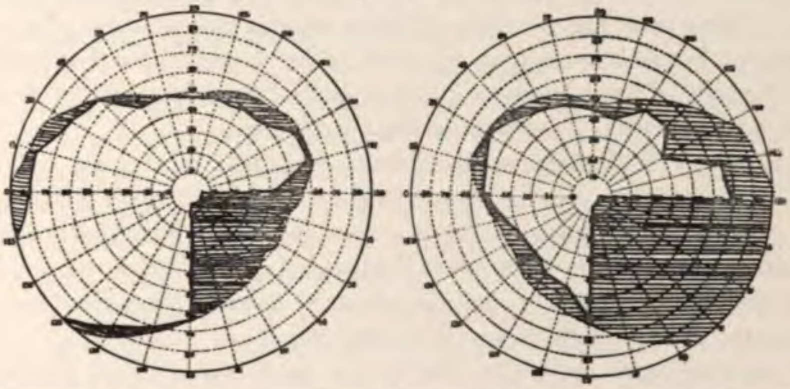
Aside from the above we may have hemianopsia associated with the most complicated conditions. Thus a complete obstruction of the entire Sylvian artery will give rise to hemianopsia, hemiplegia, and, when the left side is affected, aphasia, alexia, etc.

The mode of onset of hemianopsia varies according to the pathological cause. In diseases of the chiasm and optic tracts it is usually gradual, because the lesions appearing in this situation are nearly always of a chronic nature. Cases due to cortical diseases are not infrequently sudden in their appearance because of the possibility of the production of the trouble by circulatory disturbances (embolism and hæmorrhage). At the same time it must be borne in mind that lesions of any kind may obtain in any portion of the visual tract, each kind impressing upon the case its distinctive features.

The line of division between the normal and blind fields possesses no fixed character. It may be vertical, slanting, sinuous or indented. It may pass through the fixation-point or well within it; it may be vertical and on a line with the fixation-point; but when within a few degrees of the latter it very often curves towards the blind half, leaving the fixation-point within the seeing area. The preservation of central vision bears no relation whatever to the seat of the lesion; it probably depends upon individual peculiarities in the decussation, the fixation-point on the retina receiving fibres from both sides.

This phenomenon is susceptible, however, of another explanation, *i.e.*, the existence of a centre for central vision outside of the half-vision centres in the cuneus. Four cases bearing on this point have now been recorded. In each there was a first attack of hemianopsia, with preservation of central vision for a distance of from three to eight degrees about the fixation-point. A second attack of hemianopsia occurring, destroying the remaining half-fields, the central vision was still retained, although its acuity was considerably impaired.

FIG. 218.



Homonymous hemianopsia affecting the lower quadrants from fibro-sarcoma of the cuneus.

The course pursued by a hemianopsia is not a typical one. In some cases the defect remains stationary for years. In others it exhibits very great variations from time to time. Oppenheimer has said that an oscillating bitemporal hemianopsia is almost always of syphilitic origin. In disease of one optic tract it is not usual for the defect to extend to the uninvolved half-fields. Still, there may be concentric limitation of the latter by reason of the tract-lesion setting up inflammation of the optic nerve-fibres beyond the commissure. In hemianopsia from disease of the occipital lobe the blind half-field may extend to a line vertical with the fixation-point, but never beyond it. The fact that it has done so is significant of extension of disease to the opposite side of the brain.

The blindness is not always symmetrically distributed when the lesion is in the tract or commissure. Indeed, irregularity in the defect is here the rule rather than the exception. In disease of the occipital lobe, on the other hand, symmetry of defect is the rule. The latter symptom is therefore indicative, though not conclusively, of cortical lesion. I here append the perimeter charts of a case of lateral homonymous hemianopsia in which the lower quadrants are defective. Such cases are not uncommon. Usually they result from disease of the cuneus, the fibres from the lower half of the cuneus going to the upper half of the retina, and *vice versâ*. It is believed, and with good reason, too, that each portion of the retina finds its representation in a special portion of the cuneus. This fact is of importance in the study of scotomata, especially when the latter are symmetrical.

When hemianopsia has lasted for any length of time, it is apt to remain permanent. The only possible exceptions are those arising from syphilitic disease which has not acted destructively on the nerve-elements.

A functional hemianopsia has been claimed. There can be no doubt of its existence if we refer only to transitory attacks of this symptom in conjunction with migraine, or similar attacks unassociated with cephalic pain. These latter are probably abortive migrainous seizures. Dana has referred to lithæmia and gout as causes of hemianopsia, but I should feel very doubtful of their ability to produce such a condition without first giving rise to structural alterations in the visual fibres.

Hemiopic hallucinations have been described by Seguin, Peterson, Putzel and others. By the former author it is stated that in two out of nine cases of ordinary homonymous hemianopsia these visual hallucinations appeared in the subsequently blind field prior to the onset of the blindness. In Peterson's cases the visual disturbances existed in conjunction with mental trouble and without any associated blindness.

In certain rare cases the blindness in the half-field takes the form of a network, the remaining spots giving more or less acute vision.

Horizontal hemianopsia is nearly always due to retinal disease. Nothnagel has, however, reported a case in which he believed the trouble lay in the occipital lobes.

CHAPTER XX.

LOCALIZATION OF LESIONS OF THE SPINAL CORD.

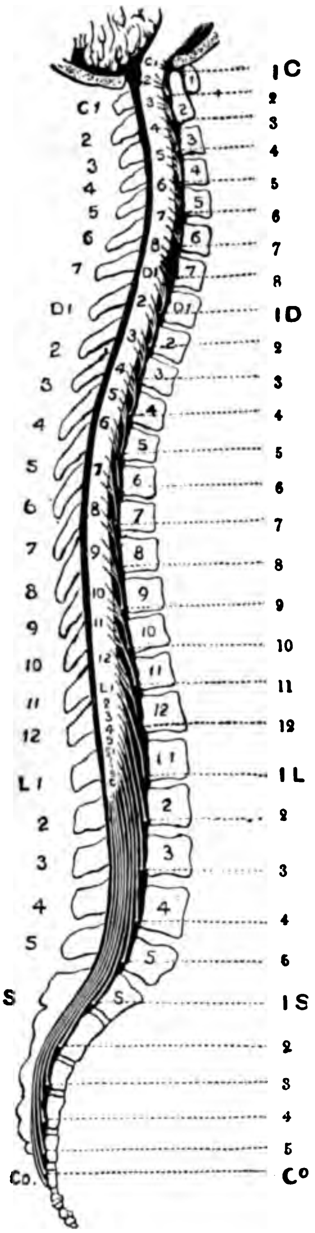
The localization of lesions of the spinal cord is of the greatest clinical importance, especially to the surgeon. In the solution of the problem there are two important factors: first, the determination of the level of the lesion; and, second, the particular portions of the cord-structure involved.

The first problem, the determination of the level of the lesion, demands a knowledge of certain important anatomical facts. The spinal cord, it must be remembered, does not extend throughout the entire length of the vertebral canal, for its lowermost extremity is on a level with the interspace between the first and second lumbar spines. For clinical study, it must be regarded as composed of a number of segments, to each of which belongs a pair of nerves transmitting motor and sensory impulses to or from definite portions of the body. The surface-guides for determining the location of these segments are the spinous processes. But it must be remembered that said processes are not opposite the cord-segment bearing the corresponding designation, nor are their apices on a level with the bodies of the vertebræ from which they spring; and, again, the spinal nerves do not emerge from the vertebral canal at points opposite the spinal segments from which they spring, for they travel some distance—sometimes long, and sometimes short—along the spinal canal before entering the intervertebral foramina. These inter-relations between the spinous processes, their vertebræ, the several cord-segments and their nerves, can be best appreciated by reference to Fig. 219 than by any wordy description. To it, therefore, the reader is referred for further information.

Next, knowledge is demanded concerning the motor and sensory functions of the several spinal segments. As to their motor functions, a good general idea may be obtained by inspection of the table presented on page 872, and which is quoted from Gowers's "Diseases of the Nervous System," vol. i., p. 252. More elaborate tabulations have been presented by other authors, but inasmuch as none of them is admitted to be exact, we must remain satisfied with the general view thus presented.

We know still less of the sensory functions of the cord than we do of the motor. Numerous authors have prepared elaborate diagrams demonstrating the areas of anæsthesia resulting from lesions at different levels. I have purposely reproduced four of these in order to show the discrepancies of the results obtained, and to demonstrate that for the present, at

FIG. 219.



Diagrammatic representation of the relative positions of the vertebral spines and bodies and the spinal segments and nerves.

least, we must rest satisfied with an approximate diagnosis; one, however, that will be sufficiently exact for all practical purposes.

The next step in the problem of spinal localization is the determination of the particular conducting tracts or portions of gray matter involved. The functions of all of these known to exist at the present time remain undetermined. Insuperable difficulties seem to exist, interfering with investigation. The motor tracts are the best understood.

The present method of studying the nervous system as composed of neurons is the most practical from a clinical standpoint. We may divide the motor structures of the spinal cord into two neurons,—the upper and lower. The upper motor neurons find their ganglion cells in the cerebral cortex, the axis-cylinder process of which pass through the pyramidal tracts, terminating at but not in immediate connection with the ganglion cells of the anterior gray horns. The lower motor neuron commences with the structures last-named and the nerve-fibre emanating therefrom, extending into the appropriate spinal nerve, and terminating in the muscles. The upper motor neurons, taken collectively, comprise the direct and crossed pyramidal tracts. By far the greater majority of them are situated in the crossed or lateral pyramidal tracts. Any lesion of them is associated with paralysis, which is attended by excessive action of the tendon and cutaneous reflexes proceeding from all spinal segments below the uppermost level of the lesion. Such paralysis, moreover, is attended by spasticity of the muscles involved.

With lesions of the lower motor neuron we have an entirely different clinical picture. This includes the ganglion cells of the anterior gray cornua and the motor nerve-roots springing from the same. The gray matter acts as a centre for and a distributor of nerve-impulses. Lesions limited to it produce symptoms referred to disturbed function of the affected segment,

FIG. 220.

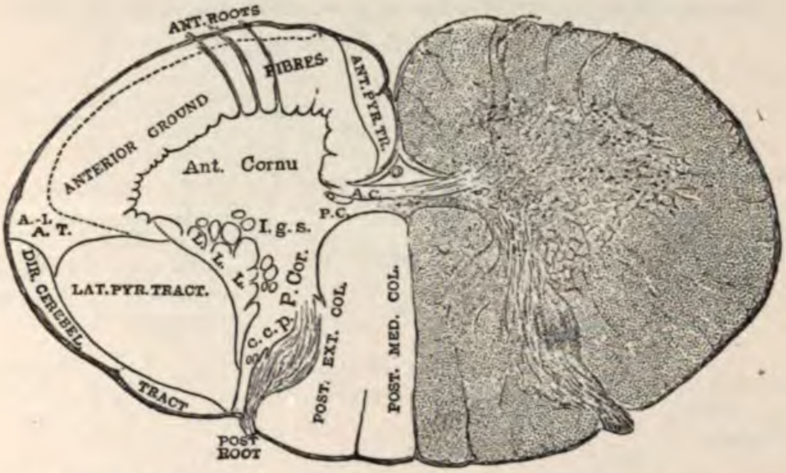
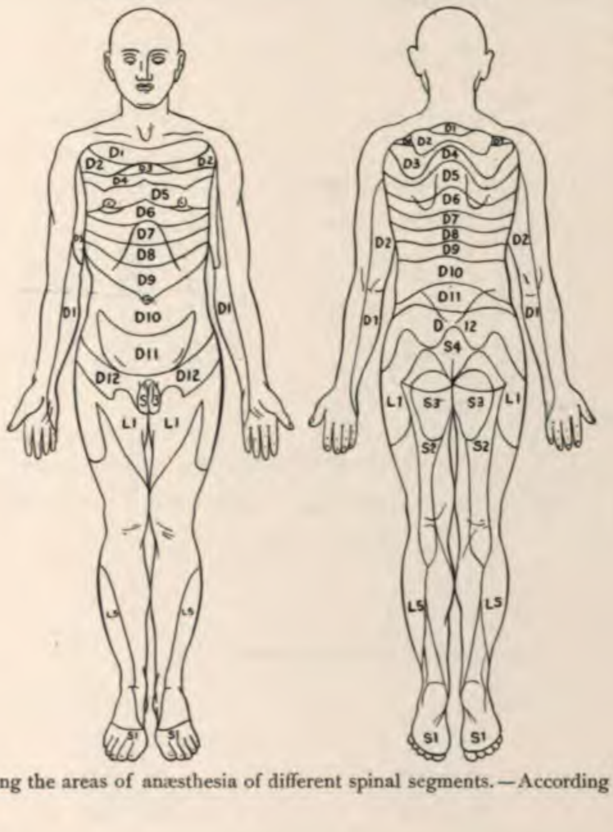


Diagram of a section of the spinal cord in the cervical region. A. C., anterior commissure; P. C., posterior commissure; I. g. s., intermediate gray substance; P. Cor., posterior cornu; c. c. p., caput cornu posterioris; L. L. L., lateral limiting area; A.-L. A. T., antero-lateral ascending tract which extends along the periphery of the cord.—After Gowers.

FIG. 221. FIG. 222.



Representing the areas of anæsthesia of different spinal segments.—According to Head.

Table showing approximate relation to the spinal nerves of the motor and reflex functions of the spinal cord.

				REFLEX.
	C. 1	Small rotators of head.		
Sterno-mastoid, upper neck muscles, upper part trapezius	*2*	Depressors of hyoid bone.		
	3	Scaleni. Lev. ang. scapulæ.		
	4	Diaphragm Pectoralis (clavicular part)	Deltoid and other shoulder muscles and sternal part of pectoralis	Dilatation of pupil.
	5	Serratus Flexors of elbow		Scapular.
Lower neck muscles, middle part trapezius	*6*	Pronators, triceps		
	7	Extensors of wrist and fingers . . .		
	8	Flexors of wrist and fingers . . . Muscles of hand		
	D. 1	Extensors of thumb		
Lower part trapezius and back muscles	*2*	Intercostals		
	3			
	4	. . . .		Epigastric.
	5			
	6	Intercostals		
	7	. . . . . .		
	8	. . . . . .		Abdominal.
	9	Abdominal muscles		
	10			
	11			
	12			
	L. 1	Ileo-psoas		Cremasteric.
Lumbar muscles	*2*	Cremaster . . . . Flexors of hip		*Knee-jerk.*
	3	Extensors of knee . . . . . Adductors of hip		
Peroneus, flexor of ankle, extensor of ankle	*4*	Extensors and abductors of hip . . .		Gluteal.
	5	Flexors of knee		*Foot-clonus.*
	S. 1	Intrinsic muscles of foot		Plantar.
	2			
	3	Perinæal and anal muscles.		
	4			
	5			

FIG. 223.

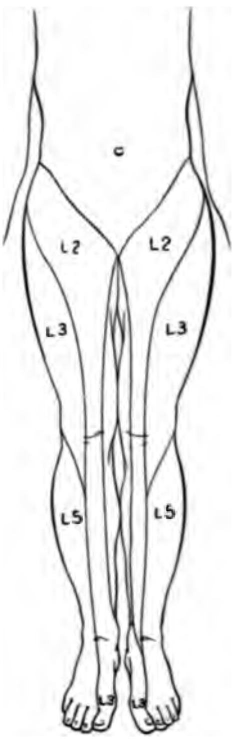
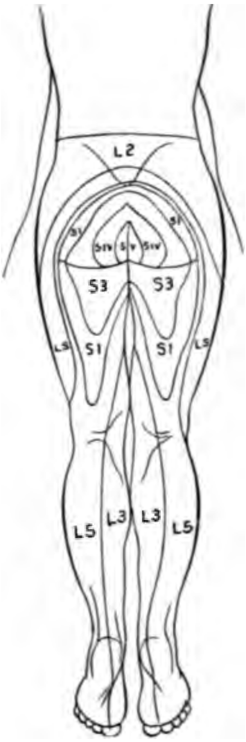


FIG. 224.



Representing the areas of anæsthesia of different spinal segments.—According to Starr.

FIG. 225.

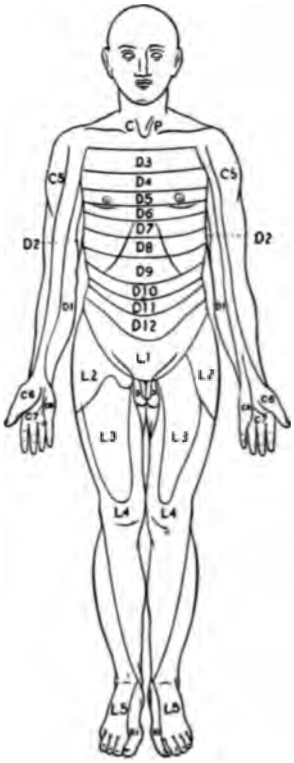
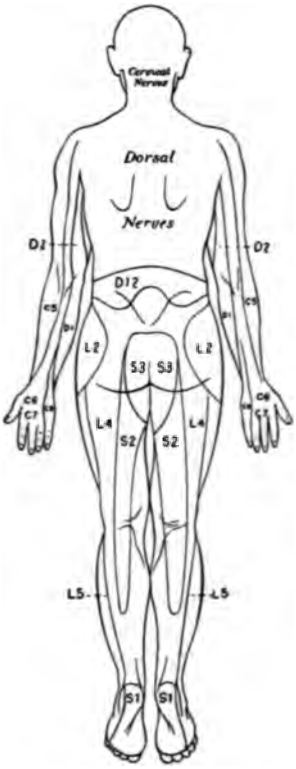


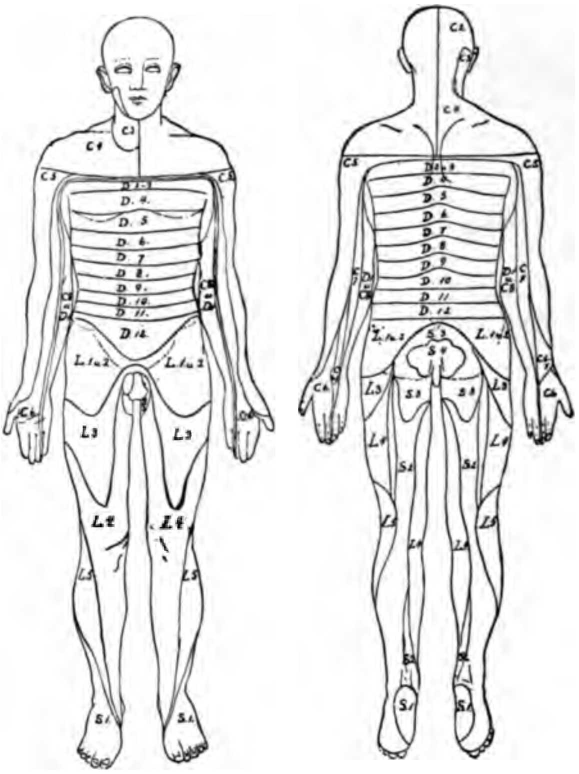
FIG. 226.



Representing the areas of anæsthesia of different spinal segments.—According to Thorburn.

and to no others. These ganglion cells exert an important trophic function, and act as reflex centres. When they are destroyed, we find, as a result, paralysis without disturbance of sensation, attended at first by flaccidity of the functionless muscles, and still later by extreme atrophy of the latter. From the beginning, the reflexes belonging to the diseased segment are destroyed. We find a good example of lesion of the anterior cornua in acute poliomyelitis anterior, or the acute spinal paralysis of infants.

FIG. 227. FIG. 228.



Representing the areas of anæsthesia of different spinal segments.—According to Oppenheim.

Knowledge as to the sensory-conducting tracts of the cord is as yet limited. We do know that the same fibres do not conduct tactile pain, temperature, and muscular-sense impulses, for all of those are not equally involved in many cases in which sensation is disturbed.

The deficient knowledge seems to have resulted from two causes. In the first place, experiments made on the lower animals are unsatisfactory as to their effect upon disturbance of sensation ; and, in the second place, in the case of pathological lesions in human beings, when both motor and sensory functions are disturbed, the latter is almost always involved to less degree than the former, and recovers more quickly.

All forms of sensation appear to be involved in lesions of the columns of Burdach. As a result of disease in this locality, we have pain, anæsthesia, and ataxia. In addition to conducting, to a certain extent, tactile sensations from the opposite posterior root, these tracts contain many associative fibres connecting different levels of the cord. Pain-sense fibres and excito-reflex fibres from the posterior roots run through these columns to reach the commissure or the anterior horns; other fibres run through it to Clark's column and to the columns of Goll.

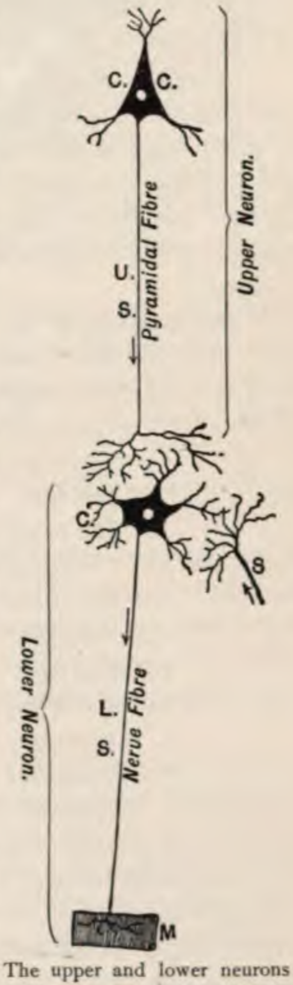
The sensibility to pain apparently lies in the gray matter near the gray commissure. Pathological evidence on this point is found in the loss of the pain-sense in cases of syringomyelia.

The paths for sensibility to temperature seem to lie near to those of the pain-sense, if we are to judge by the frequency with which these sensations are impaired in syringomyelia. The symptoms of this same disease suggest that they are far (relatively) removed from the fibres conducting general sensibility by reason of the non-involvement of their function.

The special sensation of muscles, articulations and tendinous sheaths is probably by way of the columns of Goll. As a result of lesions in this tract, we have loss of sensation of the position of the limbs, of the power of estimating weights, and of coördination of muscular effort.

The centres for the rectum and bladder are situated in the lumbar enlargement, but their exact location is not known positively. Lesions involving these centres produce incontinence or retention of fæces and urine, or both. If the lesion is situated in the centres themselves, then there is a continuous relaxation of the sphincters. In the case of the rectum, this is evident on insertion of the finger, which discovers the relaxation of the anal sphincter. In the case of the bladder, it is indicated by the constant dribbling of urine. Should the lesion be in the conducting tracts by which these centres are controlled by the brain, the involuntary defecation and micturition takes place only when sufficient contents have accumulated to act as a reflex stimulus to muscular action, when an involuntary escape follows.

FIG. 229.



The upper and lower neurons of motor path.—After Hutchinson and Rainy. C. C., cerebral cell; S. C., spinal cell; S, sensory fibre; M, muscle.

CHAPTER XXI.

THE EYE.

A STUDY of symptoms of the eye seems, at first sight, to be out of place in a text-book devoted to general medicine; but inasmuch as there is not a tissue of the organs of vision or their accessory parts that may not present important diagnostic symptoms in medical disorders, a knowledge of the eye is of the utmost importance to the general practitioner. Hence it is deemed wise to include a chapter on the eye in the present treatise.

The portions of the eye to be studied include

1. The eyelid.
2. The conjunctiva.
3. The cornea.
4. The sclerotica.
5. The iris.
6. The fundus oculi.
7. The eyeball as a whole.
8. The motor apparatus of the eyes.
9. The state of the refraction.
10. The state of the vision.

1. **The Eyelids.**—The eyelids are to be studied as to

   a. Alterations in shape.
   b. Alterations in color.
   c. Eruptions, ulcerations, etc.
   d. Motor disturbances.

(*a*) **Alterations in Shape.**—The main alterations in the shape of the eyelids relate to swelling. The most frequently encountered of these is swelling from **œdema,**—frequent because of the looseness of the subcutaneous connective tissue in this locality. Œdema of the eyelids may result from *general* or *local* causes. As a symptom of general disease it not uncommonly constitutes part of a general dropsy. In cases of chronic and acute nephritis, œdema in this locality may be the first symptom to suggest to the patient that he is ill. Such œdema is ofttimes fugitive, especially in chronic interstitial nephritis. As a rule, it is worse in the morning, and wears off in the course of the day.

Swelling of the eyelids may also be observed in anæmia, in arsenical poisoning (of which it is sometimes an initial sign), and in iodism.

The local causes of swelling of the eyelids include all inflammatory affections about the eyes, hay fever, angeio-neurotic œdema, urticaria, conjunctivitis, etc. In many instances the primary local lesion affecting the lids is secondary to some general disorder, as in the case of variola, measles, glanders, etc. The swelling of the eyelids from local lesions is ofttimes disproportionately severe to the associated symptoms. Thus it is not uncommon to observe the swelling to be so great in cases of urticaria, erysipelas of the face and scalp, rhus poisoning, etc., as to cause complete closure of the eyelids.

As to strictly local troubles, but few occasion any great degree of palpebral swelling, as a rule. Of these, ophthalmia neonatorum (gonorrhœal ophthalmia) is the most important, because of the serious results following the failure to recognize and treat it early. The swelling from catarrhal conjunctivitis and hay fever are ordinarily but slight, and inconvenient only from an æsthetic standpoint.

To the ophthalmic surgeon, œdema of the eyelids is an invaluable index of the condition of the eyeball after operation, as its presence at such time is a sure sign of infection. As long as the eyelids present a normal appearance, he has confidence that healing beneath is progressing satisfactorily.

Circumscribed swellings of the eyelids may be occasioned by styes, dacryo-cystitis, meibomian cysts, and tophi.

(*b*) **Alterations in Color of the Lids.**—The only important symptom in this connection is a darkening of the lids or the appearance of dark rings about the eyes, which are common in some persons whenever suffering from constitutional depreciation. They are not to be regarded as diagnostic of any condition; they do not even offer suggestions as to the specific direction to which inquiry should be directed.

(*c*) **Eruptions and Ulcerations of the Lids.**—The most important of these is *blepharitis marginalis*, which is characterized by swelling and redness of the edges of the lids, the affected parts being covered by numerous small whitish scales. In some cases the edges of the lids are encrusted, and, when carefully cleansed, will show small ulcerations about the origin of the lashes. Blepharitis marginalis may be produced by bad hygienic surroundings, general constitutional depreciation, exposure to smoke, dust and wind, errors of refraction, and conjunctival inflammations. Some authorities regard it as suggestive of anæmia or of the tubercular diathesis. This we doubt.

The important ulcerations of the lids include epithelioma and chancre. **Epitheliomata** appear generally in persons of advancing years, especially on the lower lid. They are characterized by ulceration, with scab formation, induration, and chronicity.

Chancre of the eyelid does not present characteristics sufficiently well-defined to make its early diagnosis an easy matter. The ulceration is small and indurated, the discharge is slight,—indeed, it may amount to nothing more than a mere moisture. The nature of the lesion is, as a rule, not recognized until the appearance of the secondary symptoms.

Tertiary ulcerations of the eyelids are of rare occurrence, and are apt to be mistaken for epitheliomata. They are, however, deeper and more extensive than the latter, and yield readily to anti-syphilitic treatment.

(*d*) **The Motor Disturbances of the Eyelids.**—The motor disturbances of the eyelids include paralysis and spasm.

Paralysis of the muscles governing the movements of the lids may produce *lagophthalmos* or *ptosis.*

By **lagophthalmos** is meant an inability to close the eye. When of paralytic origin, it is symptomatic of disease of the seventh cranial nerve, and is a prominent feature of peripheral facial palsy. Occasionally it may be due to mechanical causes, as cicatricial contraction of the skin overlying the lids, and unusual protuberance of the eyeballs, as from **exophthalmic goitre** and **retro-ocular tumors.**

Incomplete closure of the eyelids during sleep occurs occasionally with some people, especially children. It is more frequent, however, as a phenomenon of exhaustion or of exhausting disease. The eyeballs are rolled somewhat upwards ; thus only the sclerotic is exposed.

A peculiar impairment of the movements of the lids known as **von Graefe's sign** occurs in exophthalmic goitre, and is regarded as pathognomonic of that disease. It is characterized by failure of the upper lid to move in complete harmony with the eyeball when directing the gaze in a downward direction.

Another symptom of exophthalmic goitre relating to the lids is **Stellwag's sign.** It consists of enlargement of the palpebral aperture disproportionately great as compared with the exophthalmos.

Ptosis is a partial or complete inability to raise the upper lid. It is due to paralysis of the levator palpebræ superioris. It is one of the symptoms of lesion of the third pair of cranial nerves (motor oculi).

The diagnostic significance of ptosis varies with the associated symptoms. Occurring apart from other oculo-motor palsy, and without other symptoms, it may be due to injury or disease of the supra-orbital branch of the motor oculi nerve.

When associated with hemiplegia, and without evidence of involvement of other muscles supplied by the third pair, it is probably due to a cortical lesion.

When associated with paralysis of the superior rectus, it is in all probability nuclear in origin.

When associated with paralysis of all the muscles supplied by the third nerve, it is due to injury or disease of the trunk of that nerve.

But ptosis may arise from other causes than lesions of the third nerve and its branches. In **congenital** cases the defect is a muscular one, and is usually associated with other deformities of the orbit or eye.

Ptosis may be **reflex** from irritation in the domain of the fifth nerve, owing to inhibition of the centre for the levator palpebræ. The irritation, as a rule, consists of dental irritation, as caries or the extraction of a tooth. It has followed upon division of the fifth nerve.

Ptosis from **paralysis of the sympathetic** is characterized by drooping of the upper lid on the affected side associated with contracted pupil, heat and redness, and œdema of the skin and sweating on the affected side. A very similar condition is said to follow upon lesions of the corpus striatum. In ptosis from paralysis of the sympathetic, the movement of the upper lid is unimpaired. In other words, it is a **pseudo-ptosis.**

Morning ptosis occurs for the most part in weakly women. Both sides are affected. It seems to be an aggravation of the normal difficulty of opening the eyes on awakening. The condition, when pathological, usually lasts from ten to twenty minutes. During the balance of the day there is no difficulty.

Hysterical ptosis may be either single or double. It is associated with a slight spasm of the orbicularis.

Ptosis is not an unusual symptom in the course of locomotor ataxia. It is then almost invariably associated with other oculo-motor palsies.

A pseudo-ptosis may be due to disease of the eyeball. Such cases are recognized by the presence of the primary disorder and the preservation of motor function on the part of the levator palpebræ.

The spasmodic affections of the eyelids include blepharospasm, nictitating spasm, and fibrillary twitchings of the lids.

Blepharospasm is characterized by tonic spasm of the orbicularis palpebrarum of variable duration, and coming at long or short intervals. It may be due to obscure disease of the facial nerve, hysteria, or diseases of the eye of which photophobia is a prominent feature. The neurotic cases may be usually checked temporarily by pressure exerted on certain parts about the orbit, notably the point of exit of the supra-orbital nerve.

Nictitating spasm is a clonic spasm of the orbicularis, and is characterized by rapid winking movements of the eyelids. It constitutes one of the varieties of the "habit spasm."

Fibrillary twitchings are produced by momentary contractions of individual fibres of the muscles of the eyelids. They are not uncommon in states of exhaustion.

2. **The Conjunctiva.**—The objective symptoms relating to the conjunctiva include

a. Injection of the conjunctiva.

b. Discharges, which may be mucous, muco-purulent, or purulent.
c. Pseudo-membranous formation.
d. Phlyctenules.
e. Granulations (trachoma).
f. Ecchymoses.

(*a*) **Conjunctival Injection.**—Conjunctival injection is an important symptom of all inflammatory diseases of the conjunctiva. It is characterized by the formation of a coarse scarlet network of tortuous blood-vessels in the conjunctiva itself, as shown by the fact that they move with manipulation of that mucous membrane. The intensity of the injection is a safe index of the severity of the inflammation of which it is the symptom.

(*b*) **Conjunctival Discharges.**—Conjunctival discharges may be mucous, muco-purulent, or purulent. **Mucous discharge** is clear and more or less watery. It is characteristic of *simple catarrhal conjunctivitis.* It may also be produced by any irritation of the eyes, as from exposure to an irritating atmosphere, excessive use of the eye, or eye-strain.

A **muco-purulent discharge** consists of mucus in which float thick yellowish flocculi. It is present in well-developed cases of acute and chronic catarrhal conjunctivitis, and in granular ophthalmia.

A **purulent discharge** presents a thick, creamy consistence, and is of a yellowish-green color. It is found in *purulent* (*gonorrhœal*) *ophthalmia.*

A severe catarrhal conjunctivitis will sometimes produce just as marked a purulent discharge; indeed, it may be impossible to distinguish it from gonorrhœal ophthalmia by the discharge alone. The diagnosis can then only be made by the other symptoms, which, in the latter disease, are much more virulent.

Dryness of the conjunctiva is observed in collapsic states. In lagophthalmos (facial paralysis), by reason of the failure of the lids to regularly lubricate the conjunctiva, it may also occur.

(*c*) **Pseudo-Membranous Formations.**—The membranous formations of the conjunctiva include the **croupous** and the **diphtheritic.** Some claim that they are both diphtheritic. Their clinical differentiation is in many instances almost impossible. Indeed, their objective appearances are at times such that the distinctions are mainly those of relative intensity,—the bacteriological examination of the exudate alone being the diagnostic guide. **Croupous conjunctivitis** presents a thickening of the conjunctiva, with thinning of the epithelial layer of the same. The pseudo-membrane is grayish in color, and is found principally upon the lids. It may appear (though always of rare occurrence) in gonorrhœal ophthalmia, epidemic conjunctivitis, and after burns by acids, steam and scarifications. The microörganisms which have been found include the Klebs-Loeffler bacillus, streptococci, staphylococci and diplococci.

Diphtheritic conjunctivitis is due to infection with the Klebs-Loeffler bacillus, though predisposition in the shape of constitutional or local weakness is necessary as a predisposing factor. The majority of cases are secondary to faucial or nasal diphtheria, though it may appear as an idiopathic affection. The pseudo-membrane presents a gray color. It is comparatively thick, and covers the conjunctiva of the globe and lids. It is closely adherent to the surface beneath, and can only be removed with force, leaving a raw, bleeding surface. Removal is followed by rapid renewal of the membrane. The croupous membrane can be removed with comparative ease, but it also leaves a raw, bleeding surface, and is soon renewed.

(*d*) **Phlyctenules.**—Phlyctenulæ consist of small elevations, reddish in color, appearing on either the conjunctiva, the cornea or the corneal edge (*limbus corneæ*), most frequently at the latter point. They tend to break down and form small ulcerations at their apices. They heal without leaving any scar, excepting, in occasional instances, on the cornea, when the ulcer invades the substance of that structure. They are generally multiple and surrounded by a network of blood-vessels, the balance of the conjunctiva being fairly clear. They constitute the diagnostic symptom of *phlyctenular conjunctivitis* and *phlyctenular keratitis*.

(*e*) **Granulations (Trachoma).**—These consist of numerous discrete elevations, most numerous in the conjunctival fold, though existing also in great numbers upon the palpebral conjunctiva. They are characteristic of *granular conjunctivitis;* also known as *granular lids* and *granular ophthalmia*. They occur also in what has been called *follicular conjunctivitis*, a disease which, from the pathological findings, seems to be inseparable from granular ophthalmia.

(*f*) **Ecchymoses.**—Subconjunctival ecchymoses are characterized by extravasation of blood beneath the conjunctiva, and are exhibited as solid red patches, at first bright, later becoming dark, in some cases exhibiting a tendency to settle by gravity beneath the more dependent portions of the eye. They may be produced by traumatism, though more frequently they follow upon violent muscular effort, as from severe vomiting and coughing paroxysms (as in whooping-cough) and straining at stool. Ecchymoses of the conjunctiva occurring without apparent cause are supposed to indicate vascular degeneration.

(*g*) **Conjunctival Thickening.**—A thickened fold of conjunctiva, triangular in shape, with its base towards the outer part of the eyeball and its apex at the edge of or on the surface of the cornea, is characteristic of *pterygium*. In its earlier stages it is coursed by numerous blood-vessels, which converge at the apex.

The subjective symptoms of conjunctival disease are few in number and mild. They consist of sensations of heat and burning, and blurring of vision. They are of no diagnostic value.

The diseases of the conjunctiva include the following:

(*a*) Conjunctivitis, of which we have the following varieties:
  i. Simple or catarrhal conjunctivitis.
  ii. Chronic catarrhal conjunctivitis.
  iii. Phlyctenular conjunctivitis.
  iv. Purulent conjunctivitis (gonorrhœal ophthalmia).
  v. Croupous conjunctivitis.
  vi. Diphtheritic conjunctivitis.
  vii. Follicular conjunctivitis.
  viii. Granular conjunctivitis.
(*b*) Pterygium.
(*c*) Symblepharon.

(*a*) **Conjunctivitis.**—i. **Simple Catarrhal Conjunctivitis.**—Simple catarrhal conjunctivitis results almost invariably from the irritation of foreign bodies or exposure to cold. Occasionally it occurs in epidemic form, and is then due apparently to some atmospheric condition. We speak of these cases as *epidemic conjunctivitis.* We may also encounter conjunctivitis as a common complication of certain infectious diseases, especially of measles, scarlatina, and variola (*exanthematous conjunctivitis*).

It is characterized subjectively by sensations of heat and burning in the eye, obscuration of vision by the increased production of mucus, and a mild degree of photophobia. Pain occurring during the course of conjunctivitis is a sure indication of a corneal complication, probably marginal ulcer. Objectively, there are more or less marked conjunctival injection, associated in severe cases with œdema of the subconjunctival tissue (*chemosis*), mucous or muco-purulent discharge. Sometimes the eyelids are considerably swollen. The accumulation of discharge with closed lids may give rise to gumming of the edges of the lids over night. The disease usually runs a course of a few days. Sometimes it merges into a chronic conjunctivitis.

ii. **Chronic Catarrhal Conjunctivitis.**—This disease presents persistent conjunctival injection, as a rule of mild grade, associated with a slight muco-purulent discharge and redness of the edges of the lids. It is a common attendant upon the chronic varieties of rhinitis and refractive errors.

iii. **Phlyctenular Conjunctivitis.**—Phlyctenular conjunctivitis is due for the most part to some constitutional error, and is especially common among children of tubercular diathesis; hence its common designation of scrofulous ophthalmia. Its occurrence is favored by bad hygienic surroundings. Objectively, it is characterized by the appearance of phlyctenulæ upon the ocular conjunctiva, lachrymation, and in some instances the association of so-called scrofulous symptoms, as enlarged lymphatic glands, chronic muco-purulent coryza, etc. Photophobia is not prominent unless the cornea is involved.

iv. **Purulent Conjunctivitis; Gonorrhœal Ophthalmia; Ophthalmia Neonatorum.**—Acute purulent conjunctivitis is due to infection by the microörganism of gonorrhœa. The first symptoms appear in the course of two or three days after infection, and consist of a high degree of conjunctival injection, with more or less chemosis, swelling and redness of the lids, and sensitiveness of the parts to manipulation. The discharge at first is serous, and may contain some blood. It soon shows admixture of pus, and finally becomes entirely purulent. It is always profuse, accumulating within a comparatively few minutes after the affected eye has been throughly cleansed. The disease generally runs a course of three or six weeks. For some time after the subsidence of the affection there remain a swelling of the conjunctiva and chronic inflammation of the lids.

Occurring in infants (**ophthalmia neonatorum**), the first symptoms are observed two or three days after birth. They are identical with those observed in adult cases. In the latter, one eye is usually first affected; if the second eye should become involved, it is from secondary infection. In ophthalmia neonatorum both eyes are involved. The gonorrhœal ophthalmia of infants usually runs a milder course than when occurring in adult life.

Purulent ophthalmia in any event, however, is to be regarded as a dangerous affection, owing to the character of its complications. These consist of corneal ulceration and sloughing. Even panophthalmitis may ensue. The early inauguration of proper treatment almost invariably brings about good results.

v. **Croupous Conjunctivitis.**—The symptoms of this disease are those of simple conjunctivitis plus the appearance of the false membrane. The latter is seen mainly upon the palpebral conjunctiva. A clinical distinction made between it and diphtheritic conjunctivitis is that the membrane in the croupous variety does not infiltrate the tissues over which it lies. The diagnosis cannot be made positively without a bacteriological examination of the exudate. The prognosis of the affection is generally favorable. It runs a prolonged course, which may be greatly shortened by appropriate treatment.

vi. **Diphtheritic Conjunctivitis.**—Diphtheritic conjunctivitis is a much more serious affection, sloughing of the cornea, with total destruction of the affected eye, being a common sequel. The onset of the disease is sudden and its progress rapid. Swelling of the lids, with marked induration, is an early feature. The discharges are serous and blood-streaked. The diagnostic symptom is the thick, grayish, pseudo-membrane, which has already been described. It covers not only the palpebral, but also the ocular conjunctiva. Constitutional symptoms are well marked.

vii. **Follicular Conjunctivitis.**—The causes of this disease are believed to be unfavorable hygienic surroundings and depreciated constitution. It is apparently contagious, as it sometimes occurs as an epidemic in institutions and schools.

Its characteristic clinical feature is the formation of small elevations, which are due to enlargement of the lymphatic follicles of Krause, especially in the fornix conjunctivæ. The symptoms of the affection are not, as a rule, severe. They include slight thickening of the lids, lachrymation, mucous discharge, and conjunctival injection. The prognosis is generally favorable, but the disease is apt to run a slow course.

viii. **Granular Conjunctivitis; Trachoma.**—Whether or not this is the same disease as the preceding is a debatable question. The attempt has been made to base a differentiation upon the structure affected in the two diseases; but this has not been successful, as the elevations in both instances are due to enlargement of Krause's follicles. The only practical clinical distinction, then, seems to be one of severity. The etiology of granular lids is found in bad hygiene, depreciated constitution, and contagion. In the beginning, the elevations of granular ophthalmia are discrete, with some little thickening of the lids. Other than these, there is but little local disturbance. In the more advanced or acute cases, the usual symptoms of moderately severe conjunctival inflammation are associated. In still other cases considerable conjunctival thickening appears, and finally the inner surfaces of the lids are lined with a mass of lymphoid tissue. Corneal complication in the shape of opacity with vascularity (*pannus*) is common in these latter cases.

The terminal stage of the affection is atrophy of the granulations with formation of cicatricial tissue, and this, in turn, may lead to distortion of the eyelids. The disease is exceedingly chronic, and very resistant to treatment.

Diagnostic errors relating to granular lids are very common, the prevalent mistake being that of confusing it with the papilliform swellings which occur in the fornix conjunctivæ in acute and chronic catarrhal conjunctivitis. The history of the case should prevent errors.

(*b*) **Pterygium.**—The objective appearances of a pterygium have already been described. The subjective symptoms are those of conjunctival irritation. The tendency of the affection is to extend and involve a considerable portion of the cornea, becoming not only a source of inconvenience, but also endangering vision.

Pingueculæ are liable to be confounded with pterygium by the uninitiated. Their objective appearances are decidedly different, however, as they consist of a yellowish elevation or spot situated at one or the other side of the cornea—not upon it. They are especially frequent in old people. They are due to conjunctival thickening, and never cause any inconvenience other than from their appearance.

(*c*) **Symblepharon.**—Symblepharon is a cicatricial attachment of the palpebral and ocular conjunctivæ. It occurs especially after injuries, as burns; but it has been known to follow granular ophthalmia and diphtheritic conjunctivitis.

3. **The Cornea.**—The objective symptoms suggestive of corneal disease include the following:

a. Pericorneal injection.
b. Loss of transparency (opacities).
c. Ulcerations.
d. Deformities.
e. Vesicles.
f. Tumors.

(*a*) **Pericorneal Injection.**—Pericorneal or ciliary injection is suggestive of disease of the cornea, iris, or ciliary body. It is characterized by the appearance of small, straight vessels radiating from the edge of the cornea. The vessels are so fine and closely placed that they give the appearance at first sight of a diffuse, rose-colored band about the edge of the cornea. That the injection is not conjunctival is shown by it remaining stationary during manipulation of the conjunctiva.

(*b*) **Loss of Transparency of the Cornea.**—The transparency of the cornea may be lost by reason of

i. Inflammatory infiltration.
ii. The formation of cicatrices.
iii. Accumulation of pus within its substance.
iv. The arcus senilis.

i. **Inflammatory Infiltration.**—In all inflammations of the cornea there occurs an infiltration of leucocytes into the substance of the cornea, giving rise to an opacity which may be diffused, *with a steaminess of its surface*, or ill-defined and streaky. The permanence of this opacity depends upon the extent and duration of the pathological process. In order to detect corneal opacities in their earliest stage, focal illumination is absolutely necessary. A haziness that will be imperceptible to ordinary inspection will thus be made readily visible. In the majority of cases, inflammatory infiltration clears up completely.

ii. **The formation of cicatrices** follows upon severe inflammations of considerable duration and the healing of ulcers. According to their density we speak of them as *nebulæ*, *maculæ* and *leucoma*. The nebulæ are very faint; indeed, are generally visible under focal illumination. Leucoma is a dense white opacity; the macula is midway between the two in density. The surface of the cornea in these cases will be found to be normally polished, as contrasted with the steamy surface of active inflammation.

iii. **The appearance of pus within the corneal substance** indicates abscess of that structure. It is sometimes referred to as *onyx*. Abscess of the cornea can only occur from infection. It is usually secondary to ulceration.

iv. **The arcus senilis** is a narrow band of opacity along the corneal edge. As its name implies, it is frequently observed in the aged. It may occur in younger subjects by reason of degenerative changes, as beginning arterio-sclerosis.

(*c*) **Ulcerations of the Cornea.**—Corneal ulcerations present themselves in many ways. They may be superficial or deep; transparent or opaque; large or small; central or marginal; and acute or chronic. In one point only do they agree, namely, in the loss of corneal substance. They follow well-defined causes, being secondary to other diseases of the eye, especially to purulent ophthalmia, diphtheritic conjunctivitis, phlyctenular keratitis, herpes corneæ; they may follow traumatism; they may occur in conjunction with lesions of the fifth pair of nerves (*neuro-paralytic ulcer*); or they may occur in the aged, or in those whose constitution is impaired as a primary disorder. Many of them exhibit a remarkable tendency to perforation of the cornea with resulting prolapse of the iris, and in extreme cases, in which a large portion of the cornea is diseased, dislodgement and escape of the crystalline lens. They may heal without leaving any structural defects; or be followed by opacities of small or great degree, adhesions between the iris and posterior surface of the cornea (*anterior synechiæ*), or complete protrusion of the iris. They are sometimes associated with collection of pus in the anterior chamber or hypopyon.

Ulcers of the cornea have been classified as follows:

The **simple ulcer,** which is small and superficial, and exhibits no tendency to perforation. It usually undergoes healing without any permanent structural defects.

The **serpiginous ulcer,** which pursues a malignant course, tending to spread in both surface and depth. In the beginning there is an infiltration of the central corneal tissue, followed by breaking down. The margins of the ulceration show a disposition to slough. The balance of the cornea is inflamed and infiltrated. It is liable to be associated with iritis and hypopyon. The consequences of this form of ulceration are most serious, including permanent corneal opacities, anterior and posterior synechiæ, anterior staphyloma, and panophthalmitis.

The **marginal ulcer** runs a slow course, involving the cornea near its circumference.

The **herpetic ulcer,** which follows upon the rupture of a herpetic vesicle.

(*d*) **Deformities of the Cornea.**—The principal deformity of the cornea is the **conical cornea (kerato-conus).** Marked cases are readily recognized by inspection with the unaided eye. The milder degrees exhibit to the ophthalmoscope, held at a distance from the eye, an annular shadow in the red reflex. Direct ophthalmoscopic examination shows great distortion of the fundus.

Staphyloma of the cornea follows upon extensive corneal perforations, and is characterized by a marked protuberance, which consists of altered corneal tissue and the iris. It may involve part or the whole of the cornea. In color it is whitish, with bluish mottling.

(*e*) **Vesicles.**—Vesicles may appear on the cornea as a manifestation of **herpes,** or of that rare affection, **keratitis bullosa. Herpes corneæ** may occur as an idiopathic affection, in which the vesicles are confined to the cornea alone. The disease exhibits a remarkable tendency to recur. It may be a manifestation of herpes zoster, and is then associated with an herpetic eruption over the area of distribution of the nasal branch of the fifth nerve. It is characterized by intense pain and local anæsthesia. Catarrhal herpes occurs in association with catarrh of the air-passages.

In **keratitis bullosa** the vesicles are larger than in herpes. Following their rupture there is rapid healing, but recurrence is almost certain. This disease seldom occurs excepting in eyes which have been diseased, as by glaucoma or irido-cyclitis.

(*f*) **Tumors of the Cornea.**—These include fibroma, papilloma, epithelioma, sarcoma, and dermoid cysts.

The subjective symptoms of corneal disease include pain, photophobia and blepharospasm, in addition to the phenomena observed in connection with conjunctival disease. **Pain** is not apt to be very severe, excepting in cases in which there is associated lesion of the iris or ciliary body. **Photophobia** is present in all cases in which there is abrasion of the epithelial layer of the cornea. It is unquestionably the most important of the subjective symptoms of corneal disease. The **blepharospasm** is the direct result of the photophobia.

Diseases of the Cornea.—The diseases of the cornea of special interest to the practitioner may be included under the different types of keratitis, two of which are of great importance, namely: (a) scrofulous keratitis; and (b) interstitial keratitis.

(*a*) **Scrofulous keratitis,** also known as phlyctenular keratitis, occurs almost entirely among children of a strumous diathesis. It is frequently associated with glandular enlargements and evidence of bad nutrition, as anæmia. Common attendant symptoms include persistent coryza, with excoriation of the nostrils, post-nasal adenoids, etc. It is one of the more common sequelæ of measles.

Objectively, it is characterized by the appearance of one or more phlyctenulæ on the surface of the cornea, associated with more or less ocular injection. Photophobia is apt to be very well marked. Lachrymation is profuse.

While treatment brings great relief, the causes at work tend to frequent recurrences.

(*b*) **Interstitial Keratitis.**—In its initial stage, the characteristic symptom of interstitial or parenchymatous keratitis is gradually increasing opacity, rather evenly distributed over the cornea, but, on close inspection, is seen to be more dense in some parts than in others. The epithelial surface of the cornea soon becomes impaired, and then that structure presents a dull aspect. Soon the opaque cornea is invaded by blood-vessels, which, however, are so delicate and so closely situated that they give at first sight the appearance of a reddish blush to the cornea. The associated symptoms include more or less pain and lachrymation and the phenomena of the associated dyscrasia. This, in the majority of cases, will be found to be congenital syphilis.

4. **The Sclerotica.**—The only symptom of special importance referable to the sclerotica is a purplish, somewhat raised patch at a short distance from the limbus corneæ. It is significant of scleral inflammation (*episcleritis*), and is then associated with a slight degree of pain, conjunctival and pericorneal injection, lachrymation, and slight photophobia. While recovery usually follows in the course of three or four weeks, there is a remarkable tendency to recurrence. Episcleritis is nearly always due to constitutional conditions, as gout, rheumatism, syphilis, or tuberculosis.

If the inflammation involves the entire thickness of the sclerotic coat, other structures of the eye are also likely to be implicated. Pain is more severe than in episcleritis. Intraocular tension is increased, and there is great liability to secondary glaucoma. The purple patches above referred to are more extensive and better defined than in episcleritis. Other than the above, the symptoms are the same as in the preceding.

5. **The Iris and Pupil.**—The objective symptoms in this direction include alteration in the appearance of the iris, and changes in the shape, size, and movements of the pupils. So far as the iris itself is concerned, we place but little dependence upon alterations in its appearance as an aid to diagnosis, because there are other symptoms which are unquestionably more reliable for this purpose. In the only important disease of this structure, *iritis*, the iris presents a dull or lustreless appearance; but the severe pain, the pericorneal injection, *the small size of the pupil, its inactivity*, and the adhesions of the iris to the lens, constitute our reliable guides for diagnosis. Sometimes there is associated turbidity of the aqueous humor. The majority of cases of iritis are due to syphilis. Less frequently, it is an expression of rheumatism. Some few cases have been attributed to gout, diabetes, and tuberculosis.

Iritis runs an acute course, and if recognized early and proper treatment is instituted it is rare, indeed, for permanent damage to result. Improperly-treated cases result in occlusion of the pupil, with dense adhesions between the iris and crystalline lens.

The Movements of the Pupils.—While observations as to the mobility of the pupils are easily made, it is safe to say that concerning no class of symptoms does the average physician exhibit greater carelessness than he does in this connection. To be accurate, the patient must be placed so that a good light falls upon the eyes; and, in order that comparisons may be made between the two eyes, both sides should be equally well illuminated. To test the reactions of the pupils as to light, both eyes should be carefully covered, making certain that all light has been excluded. Then the covering should suddenly be removed from one eye, and the effect noticed.

Normally, the admission of light should be followed promptly by pupillary contraction. The result having been determined, the observation should be repeated upon the other eye.

It is next in order to determine the movements of the pupils during accommodation. For this purpose, the patient is requested to fix his gaze upon an object held close to his eyes, say at eight or ten inches. Normally, the pupils should contract. Then he is directed to look at some distant object, when the pupils should dilate.

The abnormalities relating to the size and movements of the pupils include the following:

Dilatation of the pupils, which we call **mydriasis.**

Contraction of the pupils, which we call **myosis.**

The qualification *stabile* is applied to mydriasis or myosis when the pupils fail to respond to light; the term *labile* is used when the pupils are movable.

The dilatation of the pupils is effected by contraction of the radiating fibres of the iris and relaxation of the circular fibres. Contraction of the pupils is produced by the converse of these. It will be readily seen, therefore, that either mydriasis or myosis may result from opposite conditions, namely, paralysis and spasm.

Pupils which fail to respond to the stimulus of light, but do move during efforts at accommodation, are known as **Argyll-Robertson pupils.** This symptom is of common occurrence in *locomotor ataxia* and *general paralysis of the insane*, and possesses a high diagnostic value.

The two pupils are not always equal in size. One only may be dilated or contracted; dilatation or myosis may exist to unequal degrees in the two eyes; or one pupil may be dilated, and the other contracted. Inequality of the pupils is known as **anicosuria.**

Sometimes the pupils react with abnormal rapidity without perceptible stimulation. In fact, contractions and dilatations occur in rapid succession. We call this *hippus.* This symptom is observed in acute meningitis, disseminated sclerosis, hysteria, epilepsy, and in some cases of tuberculous mediastinal glands.

The Consensual Reaction of the Pupils.—When light is thrown into one eye, causing the pupil of that eye to contract, observation demonstrates that the pupil of the companion eye behaves in like manner, though it received none of the light stimulus. If, in a case of blindness due, for example, to optic nerve atrophy, the affected eye be stimulated by light, there will be no pupillary movement of either eye. If the companion eye is stimulated, both pupils will exhibit the consensual reaction as in health. The refusal of the pupils to contract in case of blindness is suggestive of the lesion being in the lower portion of the visual tract. Blindness from lesion high up does not have the pupillary reaction disturbed, unless there be present some other cause for the alteration.

The semeiological aspects of the pupils have been studied by Macewen (*American Journal of the Medical Sciences*, July, 1887), and with such accuracy that the following propositions formulated by him are as true to-day as they were fifteen years ago:

"*A.* (1) When the function of the brain is in abeyance, the pupils are in a state of stabile mydriasis.

"(2) This may arise either from temporary suspension or from abolition of function.

"(3) Temporary suspension is illustrated by shock and the effect of some poisons, while the abolition of function is exemplified by extensive laceration and compression of the brain.

"*B.* (4) When the function of the brain is interfered with by conditions usually included under the term irritation, the pupils are in a state of myosis; sometimes labile, but generally stabile myosis.

"(5) This 'irritation,' or interruption of function, may be seen during certain degrees of cerebral anæmia produced experimentally, and not as a pathological result; certain amount of brain-pressure and certain stages of intracranial inflammation.

"(6) These are illustrated in persons who have suddenly lost a considerable quantity of blood (about a fifth of the whole); in the growth of intracranial tumors and the formation of sanguinolent, serous and purulent effusions; when the degree of pressure may be denominated as 'medium,' and at certain periods of meningitis and encephalitis.

"*C.* (7) The same pathological factors which cause myosis may also cause mydriasis, the degree in which these factors are present being the determining-point between the former and the latter, and not merely the particular locus in the brain.

"8. It is well illustrated by cases where the hæmorrhage is repeated and is finally pushed to syncope; in instrumental pressure, which is gradually increased until it becomes great, such as arises from tumors, blood-clots, and inflammatory products.

"*D.* (9) When the function of one-half of the cerebrum is placed in abeyance by a superficial or cortical lesion, the pupil on the same side of the lesion is placed in a state of stabile mydriasis.

"(10) This is well illustrated in cases of intracranial sanguinolent effusions consequent on injury.

"*E.* (11) When the function of one-half of the cerebrum is interfered with by some source of cortical irritation, the pupil on the corresponding side to the lesion is in a state of myosis.

"(12) This is illustrated by traumatic and pathological lesions affecting the cortex of the cerebrum.

"*F.* (13) Hæmorrhage into the pons varolii, when small, causes strongly-contracted pupils; but when it is more extensive, involving the gray matter beneath the aqueduct of Sylvius, a state of stabile mydriasis ensues.

"(14) Effusions into the lateral ventricles, when small, produce contraction of the pupils, but when the effusion is great, stabile mydriasis ensues.

"(15) Inequality of the pupils indicates a unilateral lesion or lesions.

"(16) When the lesion is cortical and unilateral the pupillary manifestations are on the corresponding side. When the basal nerves are affected unilaterally, the pupillary effect is manifested on the same side as the lesion. When the lesion is unilateral and affects the function of the white fibres of the cerebrum, the opposite pupil is generally affected. When the basal ganglia are affected unilaterally, the pupil is sometimes affected on the same side as the lesion, occasionally on the other side. In lesions of the cerebral peduncles, the pupil is affected on the same side as the lesion. Lesions in the corpora quadrigemina affect both pupils, irritation causing contraction, destruction causing dilatation and immobility. Section or destruction of one optic tract causes dilatation of the opposite pupil and blindness of the opposite eye.

"(17) Irritation of the cord, especially of the cilio-spinal axis, produces dilatation of the pupils, while destruction of the cord causes contraction. These effects are generally seen in both pupils, though experimentally, at least, they may be confined to the same side as the lesion.

"(18) The pupils are affected in the same way by lesions of the sympathetic, though in unilateral lesions it is only the pupil on the same side as the lesion which is affected.

"(19) Speaking generally, when myosis is due to a cerebral cause it indicates the earlier stages of various affections; when due to a spinal lesion, it points to a most serious paralysis, often to a destruction of the part. When mydriasis arises from a cerebral lesion it is generally present in large amount; when due to a spinal affection it indicates irritation of the part.

" Myosis occurs under the following conditions :

"(1) When a bright light acts upon the retina.

"(2) Accommodation for a near object.

"(3) Rotation of the eyeball inwards.

"(4) Local irritation or painful affections of the eyeball.

"(5) Irritation of the oculo-motor nerve.

"(6) Paralysis of sympathetic roots of lenticular ganglion or trunk of sympathetic in the neck. In paralysis of the fifth there is myosis and inflammation, passing on to destruction of the eyeball.

"(7) Paralysis of the cilio-spinal region of the spinal cord. All affections which destroy the cervical spinal marrow and intercept its conductibility produce congestion of the face and contraction of the pupils. In neurosis, which suspends or diminishes the tone of the sympathetic or spinal axis.

"(8) Encephalic congestion, such as obstacle to return of blood in the jugulars; venous congestion due to cardiac causes; active hyperæmia; plethora, fevers, pneumonia, hepatitis, etc.; when an animal is suspended by the heels; in early stages of meningitis and encephalitis; in acute mania with marked activity of the cerebral circulation; in chronic mania pupils are variable; when contracted are said to indicate supervention of paralytic dementia.

"(9) During sleep; some believe this to be due to the congestion of the cerebral vessels and those of the iris; others to the inward rotation of the eyeball.

"(10) In the early stages of cerebral tumor.

"(11) In small hæmorrhages into the cerebellum. In irritation of the cerebellum, contraction of the pupil on the same side as the lesion ensues.

"(12) Electrical stimulation of the angular gyrus frequently causes contraction of the pupil.

"(13) During forced expiration, when the eye is at the same time passive. Also generally seen during the period of apnœa in Cheyne-Stokes respiration.

"(14) Convulsions arising from meningo-encephalitis are said to be accompanied by myosis, while in convulsions due to epilepsy and in epileptiform fits they are usually accompanied by mydriasis.

"(15) When the pupil contracts on accommodation to a near object, yet does not contract to light, this indicates a lesion situated between the corpora quadrigemina and the oculo-motorius. This affection is known as the Argyll-Robertson symptom. It is seen in locomotor ataxia, and occurs generally in paralysis of the insane.

"(16) During uræmic coma.

"(17) Myotics: physostigmine, nicotine, pilocarpine, muscarine.

"Mydriasis occurs under the following conditions:

"(1) In darkness or subdued light.

"(2) Accommodation for distant objects.

"(3) Rotation of the ball outwards.

"(4) In forced movements discharged from the medulla; vomiting, swallowing, chewing, forced respiration.

"(5) Paralysis of the oculo-motor (accompanied or not by immobility of the eyeball, external strabismus, diplopia, etc.).

"(6) Destruction of the optic; amaurosis. When unilateral, associated movements continue.

"(7) Irritation of the sympathetic; powerful impression on sensory nerves; strong moral emotions, mental pain, grief, fear; neuralgia of the fifth nerve.

"(8) Irritation of the spinal cord, especially the cilio-spinal region.

"(9) Encephalic anæmia: In all cases where there is reflex contraction of the vessels of the head; when loss of blood from the body is excessive; obstruction of the carotid arteries; in thrombosis of the brain sinuses; dilatation of mesenteric vessels, when extreme; syncope, intense cold, rigors; dyscrasias of the blood, convalescence, cachectic conditions; asphyxia, epilepsy, in certain stages of these affections.

"(10) Pressure of cerebrum when great in amount, as from hæmorrhage, neoplasms, etc. In the last stages of meningo-encephalitis.

"(11) In cerebral softening. In acute dementia (œdema of cortex cerebri), observers state that the pupils are invariably dilated (Hutchinson).

"(12) In idiots the pupils are generally dilated.

"(13) During deep inspiration, generally in respiratory period of Cheyne-Stokes breathing.

"(14) Hæmorrhage into centrum ovale and into cerebral peduncles.

"(15) Ferrier produced dilatation of the opposite pupil by destructive lesion of the optic tract in the thalamus, indicative of rupture of the centripetal fibres to the irido-motor nucleus in the floor of the Sylvian aqueduct.

"(16) In hydrophobia there is mydriasis.

"(17) Mydriatics: Atropine, homatropine, duboisine, daturine, hyoscyamine, cocaine. Curare, injected subcutaneously in animals, induces in one to two hours complete paralysis of the third nerve."

Dilatation of the pupils is an important symptom of glaucoma, in which case it will be associated with concentric diminution of the visual fields, increased intra-ocular tension, and the ophthalmoscope will demonstrate cupping of the disc.

Paralysis of the accommodation, or loss of power of accommodation for near vision, is due to paralysis of the ciliary muscle. It usually takes place in conjunction with paralysis of the iris, but not necessarily so. It is most frequently observed as one of the sequelæ of diphtheria, and in association with other ocular palsies from syphilis, tuberculosis, etc.

6. **The Fundus Oculi.**—The special lesions of the fundus having important clinical relations to general medicine include retinitis albuminurica, leukæmic retinitis, retinal hæmorrhage, retinitis pigmentosa, atrophy of the optic nerve, and optic neuritis.

Retinitis depends for its recognition entirely upon ophthalmoscopic examination. Its characteristic symptom is a loss of transparency of the retina, as shown by diffused haziness or circumscribed opacities and swellings. In advanced cases there are areas of exudation exhibited as white spots, which may be confluent or discrete. Changes in the blood-vessels, when present, take the form of increased tortuosity when passing over swollen or infiltrated tissues, and are obscured in the infiltrated parts. Hæmorrhages may be present, and invade either the nerve fibre layer or the deeper portion of the retina. In the former case they exhibit frayed edges; in the latter they are more compact, rounded, and possessed of clear-cut margins. In old cases the hæmorrhagic spots are replaced by deposits of pigment. The disc may or may not give evidence of inflammation.

In **retinitis albuminurica** the characteristic objective symptom is the arrangement of a number of small white spots in stellate form about the macula. Some cases present a high degree of associated neuritis,—so great, indeed, that unless the region of the macula is examined carefully, the true condition may fail of recognition, and the so-called neuritis be accepted as evidence of the presence of a brain tumor. Retinitis albuminurica may appear during the course of any of the varieties of Bright's disease; but it is especially frequent in chronic interstitial nephritis, it being generally conceded that it occurs in about one-fifth of the cases. In the majority of cases it is to be accepted as evidence of advanced renal changes, the exceptions being found in those cases in which the nephritis is but one of the conditions attendant upon a widespread arterio-sclerosis. The majority of cases of chronic nephritis with retinitis die within six months or a year after the appearance of the latter condition. The disturbance of sight from retinitis albuminurica is very uncertain. In many instances it may be the first symptom to drive the subject to the physician, when an ophthalmoscopic examination reveals the unfortunate condition. Occurring in the course of the acute nephritis of pregnancy or scarlatina, retinitis is still of serious omen, though not a necessarily fatal sign.

Diabetic retinitis does not seem to present characteristic changes sufficient for diagnostic purposes, as in retinitis albuminurica, for a variety of retinal changes have been described as occurring in this disease. Numerous white spots grouped irregularly about the macula, usually associated with punctate hæmorrhages, but with coexistent neuritis, constitute one type. In another class of cases, owing to the association of an interstitial nephritis, retinitis albuminurica appears. Still other changes in the fundus of the diabetic include hæmorrhagic retinitis, neuritis, neuro-retinitis, and atrophy of the optic nerve. The prognostic value of retinal changes in diabetes seems to be a matter of dispute, some claiming that they are of serious significance, while others assert that the prognosis must be based entirely upon the general condition of the patient.

Leukæmic retinitis presents appearances in keeping with the condition of the blood and the well-known hæmorrhagic tendency of leukæmia. The retina is much swollen and the blood-vessels are greatly dilated. The condition of the blood causes the entire retina to assume a pale or yellowish hue. Hæmorrhagic extravasations and whitish or yellowish spots of exudation are also observed.

Retinitis hæmorrhagica, commonly spoken of as a separate variety of retinitis, may usually be classed among the other forms of retinal inflammation. Retinal hæmorrhage without coexistent retinitis may occur as the result of rupture of diseased vessels in the aged, pyæmia, septicæmia, ulcerative endocarditis, diseases of the liver and kidneys, the essential anæmias, hæmophilia, scurvy, diabetes, gout, malaria, chronic heart diseases, and glaucoma.

Functional Disturbances of the Retina.—These consist of toxic amaurosis, hysterical amblyopia, hyperæsthesia of the retina, hemeralopia and nyctalopia. Amaurosis and amblyopia may be defined as conditions in which there is loss or diminution of vision without ophthalmoscopic changes to account for the same.

Toxic amaurosis is of not infrequent occurrence as a manifestation of uræmia. It often comes on suddenly ; indeed, the patient may wake finding himself blind. While frequently associated with convulsions, it may be the single manifestation of the toxæmia. As a rule, it disappears when the function of the kidneys is restored.

Amaurosis may also result from lead, quinine and salicylic acid poisoning. Arising from the first named, it is of very rare occurrence. When lead affects the vision, it usually does so by first producing optic nerve atrophy or neuritis.

The frequency of *tobacco amaurosis* has been variously estimated. Some regard it as of very rare occurrence ; others believe it to be a not very uncommonly observed condition. It occurs especially in persons who have indulged to excess in alcohol. Chewing tobacco seems to be more injurious in this direction than is smoking. It is characterized clinically by obscuration of central vision, especially for colors, notably red and green. If not properly treated, organic changes in the optic nerve may develop.

Hysterical amblyopia may be either complete or incomplete, temporary or permanent. It is characterized by limitation of the visual fields. It is usually associated with other hysterical symptoms, although it may occur as an isolated symptom.

Retinal hyperæsthesia is symptomatic of hysteria, and of a weak state of the system generally. Strictly speaking, the term " hyperæsthesia," as applied to the retina, should convey the idea of increased acuity of vision, —a condition that is of the rarest kind. It is really applied to a state in which light produces unpleasant effects. It is to be distinguished from photophobia accompanying affections of the outer tunics of the eyes.

Reflex amblyopia has been observed in cases of irritation in the course of some of the cranial nerves, notably the fifth, and in diseases of the stomach.

Hemeralopia is a condition in which objects are seen clearly during the day, but not in the subdued light of evening.

Nyctalopia is the reverse of hemeralopia.

Optic neuritis, or inflammation of the optic nerve, may occur as an idiopathic affection, though very rarely, and as symptomatic of quite a variety of diseases, notably of tumor of the brain. In the latter affection it is especially liable to be present when the lesion involves the base; while in tumors of the convexity it is absent with a sufficient degree of frequency to make it an unreliable sign. It may also be present in other cerebral diseases, notably in meningitis, abscess, and thrombosis. It has been observed as a sequel of certain acute infectious diseases, especially scarlatina, typhoid fever, variola, etc. It is sometimes seen as an essential symptom of syphilis. Cases have originated from exposure to cold in persons of a rheumatic constitution. In women it is sometimes symptomatic of anæmia and menstrual disturbances. Quite recently, cases of chlorosis associated with optic neuritis have been reported, and incorrectly diagnosed as brain tumor, recovery or autopsy demonstrating the incorrectness of the diagnosis. Local lesions, as orbital diseases, *e.g.*, tumor, caries, periostitis, etc., explain some cases.

The diagnosis of optic neuritis depends exclusively upon the ophthalmoscopic examination. The disc exhibits an increased redness, and its borders are obscured. Swelling is next observed, and this increases until, finally, the form of the disc is lost entirely. Its location is recognized only by the point of convergence of the vessels. The arteries are usually not increased in size, and are always partially concealed by the swelling. The veins are distended and tortuous. Hæmorrhages are often observed.

Sight is not necessarily disturbed. Cases have been observed in which acuity of vision, the color-sense and the normal fields have all been preserved. Usually, however, all of these are affected. The extent to which sight is disturbed varies greatly in different cases. Very often, vision does not show marked diminution until the stage of subsidence of inflammation.

The prognosis depends entirely upon the causative factors at work.

Slight cases may subside, leaving no disturbance of vision. Severe cases may go on to absolute blindness.

Atrophy of the optic nerve may be either primary or secondary. The latter is most frequently observed after neuritis. The primary optic atrophy is most frequently observed as a symptom of degenerative affections of the nervous system, as locomotor ataxia, disseminated sclerosis, and general paresis. In *brain tumor* it is due to pressure exerted upon the chiasm or tracts by the growth itself or by intraventricular effusion. It occurs in about one-twelfth of the cases of *locomotor ataxia*, although ophthalmologists are inclined to assign it a higher percentage, probably because the cases not attended by eye-symptoms do not come under their observation. In the majority of cases it is an early symptom, appearing before the characteristic features of the disease become obtrusive. It is a curious fact that those cases of ataxia ushered in by optic-nerve atrophy do not progress to the advanced stage of helplessness observed in typical tabes. The optic-nerve atrophy of *general paresis* is usually an early symptom. Unlike locomotor ataxia, the cases in which it occurs pursue the same course as do those without this symptom.

Primary optic atrophy is occasionally observed. Some of these cases seem to depend upon family predisposition. As a rule, they do not progress to complete blindness.

Orbital lesions sometimes exert a deleterious effect upon the nutrition of the optic nerve, and atrophy of that structure is a logical result.

Internal hydrocephalus is sometimes associated with optic-nerve atrophy, due, in all probability, to the pressure of the effusion upon the chiasm and tracts.

The diagnosis of optic-nerve atrophy is based upon the ophthalmoscopic findings. The disc presents a bluish or grayish-white hue, the vessels are more or less reduced in size, and the lamina cribrosa *may* become plainly visible.

In post-neuritic atrophy the calibres of the vessels are greatly reduced. The margins of the discs are obscured; the blood-vessels are tortuous, and exhibit white streaks of connective tissue along their course. The disc itself may present a fine mesh-work of blood-vessels; in this respect the condition presents an important difference from primary atrophy.

The disturbance of vision from optic-nerve atrophy is always well-marked, and usually progresses, despite treatment, to complete blindness. It generally begins with contraction of the visual fields, or sector-like scotoma. The color-sense, in particular, shows impairment.

7. **The Eyeballs.**—The special symptoms in this connection include exophthalmos and enophthalmos. **Exophthalmos** occurs as a symptom of orbital growths or retro-ocular œdema, and is one of the characteristic symptoms of exophthalmic goitre. As a symptom of retro-ocular œdema,

it is apt to be due to thrombosis of the superior longitudinal or cavernous sinus. In exophthalmic goitre it is nearly always bilateral.

Enophthalmos, or recession of the eyeballs, is observed in conditions of collapse and in wasting diseases.

8. **The Motor Apparatus of the Eyes.**—The cranial nerves through which the eyes receive their motor power are the *fourth*, or *patheticus*, which supplies the superior oblique muscle; the *sixth*, or *abducens*, which goes to the external rectus; and the *third*, or *motor oculi*, which supplies the remaining muscles, both external and internal. Paralysis of any one of the ocular muscles produces certain well-defined symptoms, differing in character, however, according to the special muscle affected. It will conduce to a readier understanding of what is generally regarded as a very intricate subject, I believe, if I first detail the symptoms of paralysis of the motor nerves of the eye in general, leaving to the last of this section a description of disease of special nerves and muscles.

Etiology.—Paralyses of the ocular nerves may be congenital or acquired. The great majority of the latter cases are syphilitic in origin, the remainder being rheumatic or secondary to some infectious disease, especially to diphtheria. Some few result from organic brain disease, as tumors of a sarcomatous or tuberculous nature. In childhood, the majority of cases of ocular paralyses are of tuberculous origin. Occurring as a symptomatic condition, these palsies are observed during the course of locomotor ataxia, disseminated sclerosis, general paralysis of the insane, and some other central organic affections.

Pathology and Morbid Anatomy.—A complete exposition of the pathology of oculo-motor paralysis involves a far more lengthy explanation than space will permit. It is only possible here to refer to the subject in the most general way. Ocular paralysis may result from focal or general lesions of almost any character. If the former, the damage may be cortical, in the nerve nuclei themselves, in the association fibres, in the fibres between the nuclei and point of emergence of the nerve from the brain, in the course of the nerves at the base of the brain or through the foramina or within the orbit. Syphilitic and tuberculous processes affecting, as they are especially apt to do, the basal meninges, are liable to interfere with the function of these nerves, all of which travel an unusually long and exposed course before leaving the cranial cavity. In some cases we find small foci of degeneration damaging the nuclei or the various systems of fibres. Still others are of very uncertain pathological nature, certain rare transient and recurring paralyses of the third pair of nerves being deserving of especial mention in this connection.

Symptoms.—The one cardinal symptom of paralysis of an ocular nerve is *strabismus*—a strabismus that presents certain points of distinction from the ordinary concomitant squint, so common as the result of refractive errors. The paralytic squint is marked by defective movement of the eyeball in the direction of action of the affected muscle or muscles, diplopia, false projection of the visual field, and greater secondary deviation of the sound eye. Each of these conditions requires explanation.

(*a*) **Defective Movement in the Direction of Action of the Paralyzed Muscle or Muscles.**—In the great majority of cases of paralysis of eye-muscles, the deviation of the eye is patent to even the most careless observer. The general appearance of the deformity does not differ, however, from that of concomitant squint until certain tests have been applied. By holding an object before the patient, and moving the same in different directions while the patient follows it with his eyes, the activity of the muscles of these organs may be readily judged. Care must be taken, however, that the patient does not move his head, even in the slightest degree, while this observation is being made.

(*b*) **Diplopia.**—This is always present in paralytic squint of comparatively recent onset. In old cases, the patient learns to disregard the false image and sees only the true. In many such, however, the diplopia can be restored by placing a red glass before one eye, preferably the sound one. The relative positions of the true and false objects will vary according to the extent of the lesion and the muscle involved. If the paralysis is but a very slight one, the separation of the images is correspondingly small. The false image may be above or below or to either side of the true one, or it may be inclined at an angle to the true image, and still be in any of the above-named relative positions.

It is customary to speak of diplopia as *simple* or *homonymous* and *crossed diplopia*. Simple or homonymous diplopia is that variety in which the right-hand image is seen by the right eye and the left-hand image by the left eye. In crossed diplopia the right eye sees the left-hand image and the left eye the right-hand one. Homonymous diplopia is produced by convergent squint and crossed diplopia by divergent squint. This statement is a simple one, but can readily escape the memory. For our guidance we have a rule formulated by Gowers, and this is: "*When the axes of the eyes, if prolonged, cross, then the images are not crossed; and vice versâ.*"

In order to distinguish as to which eye each image belongs, it is only necessary to place a red glass before one eye, which will color the corresponding image.

To determine the extent of the paralysis and its variations from day to day, several methods are at our disposal. We may measure it by means of a little instrument consisting of an arc with degrees marked on it; or by means of a perimeter determine the field over which the patient has binocular vision of the test object. Lastly, we may determine the degree of the prism required to overcome the double vision. In applying the latter test, it should be remembered that the base of the prism should be placed in the direction of the weakened muscle.

A patient with diplopia assumes peculiar attitudes of the head, that he may overcome the visual error in part, at least. So characteristic is this that a skilled observer can sometimes by this alone determine the trouble. The head is carried in the direction of the action of the paralyzed muscle. Thus in paralysis of the right external rectus the head is apt to be turned to the right, because in that position the diplopia is at a minimum, if not entirely obviated. Looking towards the left will intensify the symptom.

(*c*) **False Projection of the Visual Field.**—By this expression we mean to convey the idea that the ability of the patient to judge correctly of the position of objects is impaired. In health, we are enabled by our visual apparatus to judge precisely of the position of an object before us, and reach for it correctly. We do this by estimating the amount of muscular exertion required by the eyes in bringing their axes to bear on it. This is done unconsciously, of course. If the eyes are at rest in raised position, and an object is in front of it, we know that the object is in front of our face. If that object is moved to the right, we can judge, by following it with our eyes, of the extent of that change. If, however, the right external rectus is paralyzed and we attempt to follow the object, we require greater innervation than is normally given to that muscle; and, judging by the extra exertion thus made, we are led to locate the object much further to the right than it really is, and make a reach for it accordingly. This false interpretation of position leads to a not infrequently experienced symptom in ocular palsies, namely, vertigo. To overcome this, the patient is often led to close the affected eye.

(*d*) **The Secondary Deviation of the Sound Eye is Greater than the Primary Deviation of the Affected Eye.**—If, in a case of paralytic squint, the patient fixes with the sound eye, a deviation will be observed in the affected one. This is the primary deviation. If now the sound eye is shielded, and the patient be made to fix with the one affected, the former deviates, and it will be observed that this deviation is greater than that observed in the affected eye. This is the secondary deviation. These phenomena are easily explained. In health, an equal amount of nerve-force is sent to each eye when the subject is required to fix with either. If, in a case of ocular paralysis, the patient fixes with the well eye, only the normal amount of nerve-force is expended in making the movement, and goes to each organ. The diseased eye exhibits a deviation corresponding to the intensity of the paralysis. To fix with the affected eye requires an expenditure of much greater than the normal force, and this is transmitted likewise to the normal one. Consequently, acting under its influence, it exhibits far greater deviation than did its fellow.

It would seem that with a little care and experience the recognition of ocular palsies is an easy matter. This is true in the majority of cases when seen early. But circumstances will arise in which the most expert will be utterly unable to determine the precise condition present. Complicating factors creep in, and, as it were, upset every possible calculation. Several muscles may be affected to unequal degrees, in some the paralysis being complete, in others partial. This offers a difficult problem for solution. Sometimes paralysis sets in in cases in which the muscular equilibrium had previously been affected. This muscular insufficiency is reflected in the character of the diplopia. Sometimes the trouble appears in patients whose eyes have very unequal visual powers, and the new lesion affects the better eye. The patient is then obliged to use the latter for fixation, and the amblyopic eye is in a state of secondary deviation. The diagnostic danger in such a case is that of considering the sound eye the paralyzed one.

In old cases the unaffected muscles often undergo secondary contractures, producing almost any possible deviation. This impresses itself on the diplopia, the *modus operandi* of which becomes almost unfathomable.

Paralysis of the Individual Oculo-Motor Nerves.—Third Nerve.—This nerve supplies all the muscles of the eye with the exception of the external rectus muscle and the superior oblique. All the movements of the eye are therefore abolished, with the exception of abduction and rotation downwards and inwards. The power of accommodation is lost, and the pupil is moderately dilated and does not react to light.

When the paralysis is complete, there is drooping of the upper lid (ptosis), and deviation of the eyeball outwards and downwards. The diplopia is crossed. The two images are on the same plane when the object is horizontal with the eyes. When, however, the object is below, the false image appears below the true; and when above, the true image occupies the lower position. Sometimes, when all the eye-muscles are paralyzed, there is a slight protrusion of the eyeball (*exophthalmos paralyticus*).

Paralysis of the levator of the upper lid occurs in quite a variety of conditions. It is probably observed as a symptom of locomotor ataxia or hysteria more frequently than under other circumstances. It may also result from nuclear disease, in which case it is apt to be associated with paralysis of the superior rectus. It has been seen as a congenital condition, these cases being practically incurable. A few examples arise from cortical lesion. A transient drooping of the upper lid is observed in some delicate women, especially in the morning.

Paralysis of the superior rectus gives rise to a deviation of the eye downwards, with slight divergence owing to the unopposed action of the inferior oblique. The diplopia is aggravated when the patient attempts to look upwards. The false image is seen above the true, and is tilted, in the case of the affection of the left eye, to the patient's right.

Paralysis of the internal rectus produces divergent strabismus. The diplopia is crossed.

In **paralysis of the inferior rectus** the affected eye deviates upwards and slightly outwards. The relative positions of the images is the exact converse of that given above for paralysis of the superior rectus.

In **paralysis of the inferior oblique** the defect in movement is upwards and inwards. It occurs very rarely as an isolated condition, and when associated with other ocular palsies is difficult of detection.

Fourth Nerve.—This supplies the superior oblique muscle, paralysis of which causes a loss of movement downwards and inwards. The strabismus is convergent, and is observed especially when the patient looks downwards, there then appearing a simple diplopia. This condition is very liable to be mistaken for paralysis of the superior rectus. In the latter case, however, the loss of movement is downwards and outwards, and the diplopia is of the crossed variety, and is aggravated when the eyes are directed upwards.

Sixth Nerve.—Paralysis of the sixth nerve causes loss of power in the external rectus, with a convergent squint and homonymous diplopia.

Diagnosis of the Seat of the Lesion.—Cortical lesions do not produce ocular palsies other than ptosis, and even this is rare from such a cause. When the lesion is nuclear, a number of muscles are generally affected. One external muscle after another is affected, leaving the internal muscles unaffected. Nuclear involvement of but one muscle is almost always confined to locomotor ataxia, disseminated sclerosis, etc. A fascicular paralysis, that is, one dependent upon damage between the nuclei and the point of emergence from the brain, is exhibited by alternate hemiplegia. In the case of the third nerve, the oculo-motor paralysis on one side, and that of the face, arm and leg on the other; in the case of the abducens, there is an alternate paralysis also, the parts affected being the external rectus and face on one side, and the arm and leg on the other. When the nerves are involved in their course at the base of the brain, one cranial nerve is attacked after another; the invasion of the trigeminus is exhibited by neuralgia as an introductory symptom; blindness appears in one eye, without ophthalmoscopic changes. When the lesion is in the orbit, the associated symptoms must decide. Local pain, exophthalmos, optic neuritis, etc., are suggestive.

Ophthalmoplegia.—This is a term first employed by Jonathan Hutchinson to designate a progressive paralysis of all the eye-muscles, and dependent upon nuclear degeneration; hence it has been spoken of as *nuclear ophthalmoplegia*. When the external muscles of the eye only are affected, it is called *ophthalmoplegia externa;* when limited to the internal muscles, *ophthalmoplegia interna;* and when all the muscles are involved, *total ophthalmoplegia*.

Nuclear ophthalmoplegia has been observed most frequently as one of the concomitants of locomotor ataxia, general paralysis of the insane, and progressive muscular atrophy. Many cases are of syphilitic origin. It sometimes follows diphtheria. The prognosis is unfavorable.

The prognosis of ocular paralyses must depend upon their duration and cause. Those accompanying locomotor ataxia nearly always recover, though they are liable to relapse. Nearly all the syphilitic and rheumatic cases recover under suitable treatment. Cases dependent upon degenerative changes or tumors other than gummata are of ill omen.

Insufficiency of the ocular muscles has sprung into prominence of late years as an important cause of nearly all the functional ailments to which flesh is heir. To it have been attributed insanity, epilepsy, chorea, hysteria, and nerve prostration; and curious and tragic have been many of the tales related by patients who have submitted to graduated tenotomies for relief. As a matter of fact there are a few cases in which muscular insufficiency does produce considerable mischief; but in the majority of instances in which it occurs it is a symptomatic condition dependent upon the deteriorated constitution of the patient, and is in no sense the cause thereof.

For the quick detection of muscular insufficiencies, the best instrument is the **Maddox prism,** which can be obtained at such a low price as to place it within the reach of every practitioner. It consists of a round metal disc, in the centre of which is a long slit in which is set a colored (red) glass rod. The prism is held before one eye, and the patient is instructed to look with both eyes at a lighted candle. If the muscular balance is normal, there will appear a long red band running at right angles to the axis at which the prism is held, and intersecting the light, which will present the usual appearance as observed with the naked eye. If, on the other hand, there is muscular insufficiency, the red band will not intersect the light, but will pass to one or the other side of it, according to the particular muscles which happen to be weakened. The only exception to this statement is in case the prism is held in a horizontal position, when the band is commonly slightly to one side; but the difference is very slight.

Spasmodic Affections of the Ocular Muscles.—These include spasmodic squint, nystagmus, irregular movements of the eyeballs, spasm of accommodation, and conjugate deviation of the head and eyes.

Spasmodic squint occurs most frequently in conjunction with lesions producing irritation at the base of the brain. The deviation of the eyes is noticeable even when those organs are at rest in the mid-position. This form of squint is sometimes observed as a reflex from abdominal irritation and in chorea. It very commonly produces diplopia. As a rule, it is not of long continuance.

Spasm of the ocular muscles also occurs in hysteria, especially during the convulsive seizures. The movements are convergent, never divergent. Sometimes the spasm is paroxysmal. In such cases it comes on suddenly, lasts for a few seconds or minutes, and then as suddenly disappears. The full significance of such cases is not understood.

Nystagmus consists of rhythmical oscillations of the eyeball. The movements may be lateral, vertical, or rotary, the former being the most common. Their rapidity varies greatly in different cases, and at different times in the same case, the extremes being sixty and two hundred per minute. Usually the movements of the eyes in one direction are more rapid than on the return. In many cases the nystagmus is observed only when the eyes are turned in certain directions.

The causes of nystagmus are found, first, in local conditions of the eyes, and, secondly, in diseases of the nervous system. Ocular changes producing this condition are opacities of the cornea and lens, and degenerative diseases of the choroid and retina. These are operative only when occurring in infancy. Nystagmus never arises from errors of refraction. In albinos, nystagmus is very common. These defects were combined in no less than four children of one family under my care.

The nervous diseases of which nystagmus may be a symptom are many and varied. They consist mostly of chronic degenerative diseases, as disseminated sclerosis, Friedreich's ataxia, meningitis, meningeal hæmorrhage, thrombosis of the cerebral sinuses, tumor, and local softenings. Intracranial tumor is especially liable to give rise to this symptom when affecting the cerebellum. Lesions in the pons, optic thalamus and corpora quadrigemina have also caused it.

In some cases the movements may be so slight as to escape observation. In such they can be made more prominent by directing the patient to look slightly upwards.

The pathology of nystagmus is not well understood. It is known, however, that it is proof positive of organic disease.

Irregular movements of the eyeballs occur in association with ocular palsies, muscular insufficiencies, refractive errors, labyrinthine disease, chronic hydrocephalus, meningitis, and hysteria.

Conjugated deviation of the head and eyes consists of a movement of the head and eyes to one side, this being accomplished by the external rectus of one eye and the internal rectus of the other and the sterno-mastoid muscle on the side opposite to the direction of the movement. The diseases in which it is most frequently observed are cerebral hæmorrhage, brain tumor, and meningitis. It may be either paralytic or spasmodic, its nature being readily recognized by the condition of other portions of the body. It is very common in the early stages of hemiplegic seizures. That the position of the head in such cases is not due to gravity is shown by the fact that some effort is required to bring the head to the median line, and as soon as it is released it returns to its abnormal position.

The direction of the deviation will vary according to the seat and nature of the lesion. When destructive (paralyzing), a lesion above (in the hemispheres) causes deviation towards that side, and away from the paralysis; the patient attempts to look at the lesion, as it has been expressed in medical literature. With a basilar lesion of the same character, the deviation is in the direction of the paralyzed side. When the condition is one of convulsion, the opposite to the above statement obtains; in cerebral lesions the deviation is towards the side of the convulsed extremities; and in those of the mesencephalon, towards the side of the lesion. Some *apparent* exceptions to these observations have been reported.

Spasm of accommodation is one of the manifestations of eye-strain. It is characterized by temporary blurring of distant vision. It is especially liable to occur when the eyes are irritated or fatigued.

9. **The State of the Refraction.**—The determination of the state of refraction is of the highest importance, for it is not infrequent that defects in this direction constitute the cause of numerous reflex disturbances, especially, however, of headaches. It may happen, in many of these cases, that the patient experiences no symptoms whatever to suggest the eyes as the *casus mali.* It is necessary, in all suspected instances, to go over the state of the refraction systematically. A description of the necessary procedure is out of place in this work, so the reader is referred to standard works on ophthalmology for the necessary information.

10. **The Vision.**—The testing of the visual power involves the determination of the acuity of the sense of sight, and the extent and defects of the visual field. The acuity of vision is determined by certain test-types standardized for normal vision at different distances. Thus, the "twenty" type should be plainly legible at twenty feet, the "ten" type at ten feet, etc. In recording the result we make use of a fraction, the denominator of which is the distance at which the letters should be read, and the numerator the distance at which the patient is able to read them. Thus, if the "twenty type" is read at ten feet, the record of vision for that case is $\frac{10}{20}$. Each eye should be tested separately, care being observed that the one not in use is well covered.

The acuity of vision may be impaired by any of the numerous diseases of the eye, with the exception of those involving the conjunctiva. Even the latter may cause a temporary blurring of sight, owing to the accumulation of mucus over the cornea.

For determining defects in the visual fields the perimeter is an important instrument, though rough observations may be made by other methods. (*Vide* paragraphs on Hemianopsia.) The defects in the visual fields include hemianopsia, scotomata, concentric diminution of the fields, and sector-like defects.

The nature of the defects in hemianopsia has already been explained.

By **scotomata** we understand island-like defects in the visual fields. They are observed in circumscribed lesions of the fundus oculi, *e.g.*, chorhoiditis. Scotomata centrally situated are found in cases of retro-bulbar neuritis.

Sector-like defects consist of wedge-shaped deficiencies in the visual fields, their bases being peripherally situated. They are found in atrophy of the optic nerve, occlusion of a branch of the retinal arteries, and in detachment of the retina.

CHAPTER XXII.

THE EAR.*

Symptoms of Ear Disease.—The symptoms which are suggestive of disease of the ears include the following:

1. Deafness or impairment of hearing.
2. Tinnitus aurium.
3. Pain.
4. Vertigo.
5. Discharge.

The above may be said to be the common or ordinary indications of ear disease.

As **uncommon or occasional ear symptoms** we may have:

1. Fullness and pressure in the ears.
2. Sensation as of something moving in the ears.
3. Resonance of the voice.
4. Itching and soreness of the ears.
5. Double hearing.
6. Paracusis loci, *i.e.*, inability to tell from which direction sounds come.
7. Sensations of breathing sounds in the ears, or flapping sound during deglutition, and other symptoms resulting from undue perviousness of the Eustachian tubes.

1. **Deafness or Impairment of Hearing.**—The manner of onset of defective hearing, together with its symptomatic associations, offers valuable diagnostic suggestions, as follows:

Impairment of hearing, if recent and moderate, and not associated with pain, is suggestive of catarrh of the Eustachian tube, mild catarrh of the tympanum, or impacted cerumen.

Sudden impairment of hearing is suggestive of plugging of the external auditory meatus, as with cerumen.

Slowly progressive deafness with tinnitus, of changes in the tympanic cavity, or, in the case of old people, of auditory nerve sclerosis.

Sudden loss of hearing following mumps, or cerebro-spinal fever, of auditory nerve lesion.

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* For the data comprising the information in this chapter, the author is indebted to Dr. C. M. Thomas.

Sudden deafness following scarlatina, measles, small-pox, typhoid fever, or grippe, of middle-ear involvement.

Very rapidly increasing deafness, with no other symptoms, of syphilis of the auditory nerve.

Very sudden deafness following extreme vertigo and nausea shows labyrinthine lesion, probably hæmorrhagic.

Sudden deafness following head injury, with the appearance of blood in the canal, rupture of the membrana tympani.

Sudden deafness following head injury, when a flow of serum appears in association with or after the bleeding, suggests fracture of the base of the skull or auditory-nerve lesion.

Paracusis of Willis, *i.e.*, the ability to hear better in a noise, indicates middle-ear deafness of the chronic non-suppurative variety. No suitable explanation for this phenomenon has been offered.

Paracusis loci, or the inability to locate auditory sensations, has received no explanation, clinical or otherwise.

2. **Tinnitus Aurium.**—By " tinnitus aurium " is meant the appearance of subjective sounds in the ears or head. As described by patients, these sounds include ringing, hissing, buzzing, roaring, and numerous others. Sometimes they are heard only during perfect silence; only while the patient is suffering from an acute naso-pharyngeal catarrh; or at times when the patient is experiencing general ill-health. Tinnitus is very uncommon in the ear troubles of children.

It may be associated with any of the diseases of the ear. It is, however, more common in chronic non-suppurative otitis media. It is frequently associated with the so-called auditory-nerve deafness. It may be due apparently to a variety of causes, but increaseor diminution of labyrinthine tension, by reason of which alterations in pressure on the auditory-nerve ends results, is probably the actual cause. This may arise either directly or indirectly, through changes in the tympanic mucous membrane and its vessels and ossicles.

A tinnitus may result from alterations in the general vascular pressure in a variety of diseases, including under this head chronic interstitial nephritis, heart diseases, anæmia, and overdosing with quinine, salicylate of soda and alcohol.

Among other local lesions playing the parts of etiological factors are fluid in the middle ear, aneurysm of the onlying vessels of the tympanum, and foreign substances in contact with the membrana tympani.

A variety of sound sometimes complained of by patients is snapping, clicking or crackling.

As a rule, such noises are purely subjective; occasionally, however, they are perceptible to the examiner. They are believed to be due to spasm of the palatal muscles acting upon the Eustachian tubes, separating its walls, which may have become adherent by reason of the presence of a glutinous discharge. Some cases have been attributed to spasm of the tensor tympani, or of the stapedius muscle.

Tinnitus may exist as a manifestation of general nervous instability. The patients in these cases are loud in their expressions as to their sufferings, declaring that, if not relieved, they will surely go crazy; and they sometimes keep their promise. That such should be the case is not surprising, for the subjective noises are, in reality, the first sign of a disturbed mental balance. In cases pursuing a less unfavorable course it may be regarded as purely hysterical. Cases of this character should not be confounded with ordinary tinnitus from local causes, in which the associated nervousness is the result of temperamental states, the patient being intolerant of any slight ailment.

3. **Pain; Otalgia; Earache.**—The ordinary causes of pain in the ear include diffuse or circumscribed inflammation of the auditory canal, inflammation of the middle ear or otitis media acuta, inflammation of the Eustachian tube with closure, inflammation of the mastoid process (mastoiditis), and as a reflex symptom, from diseased teeth, sore tongue, rheumatism, etc.

The pain from inflammation of the auditory canal is aggravated by pressure within the canal or on the tragus, or by traction upon the auricle, or by a chewing motion.

The pain of inflammation of the middle ear is aggravated by such acts as sneezing and blowing the nose, and by pressure below the lobule of the external ear.

The pain in inflammation of the Eustachian tube with closure of that passage is the result of the marked retraction of the membranum tympani, dependent upon the rarefaction of the air in the tympanum. Pain from this cause is sometimes severe. A good example of it is found in earache in the morning following the excessive indulgence in tobacco the night before. Such pain disappears very promptly on inflation by the Valsalvian or Politzer method.

Pain is usually a prominent feature of inflammation of the mastoid. It is usually aggravated by pressure over the base or the apex of that prominence.

Pain occurring during the course of chronic suppurative disease of the tympanum suggests either the damming up of discharge or the supervention of or increase in already existing caries of the bones.

Pain arising from diseased teeth, throat inflammations and rheumatism is to be diagnosed by exclusion.

4. **Vertigo.**—The vertigo of ear disease is generally attributed to irritation or pressure on the membrana tympani or the contents of the drum cavity, thereby increasing the labyrinthine tension ; or to changes in the labyrinth itself, as exudation and hæmorrhage. It is rarely observed in the ear diseases of childhood, excepting in connection with or following cerebrospinal fever. Clinically, we note the occurrence of auditory vertigo in the course of so-called Menière's disease, the presence of inflammatory exudate in the tympanum, adhesions in the ossicular chain, occlusion of the Eustachian tube, and in the course of certain manipulations about the ear, as syringing and Politzer inflation. It sometimes happens that the association of vertigo with aural disease is really dependent upon a sequential involvement of the brain. Such cases call for the utmost care in their study and management.

The actual relation between ear disease and vertigo is a mooted point,—some claiming that all vertigoes are of aural origin, while others are satisfied with stating that "vertigo is occasionally due to pathological conditions in the ear."

Because of the intimate relation between this symptom and ear disease, vertigo is best studied in the present connection.

Clinically, vertigo may arise from the following conditions:

a. Organic disease of the central nervous system.
b. Disease of the circulatory apparatus.
c. Epilepsy.
d. Hysteria.
e. Neurasthenia.
f. Disease of the special senses.
g. Peripheral irritation.
h. Toxæmia.
i. Gerlier's disease.
j. Conditions in which the causes in the present state of medical knowledge are undiscoverable. Such cases may be designated "essential vertigo."

(*a*) **Vertigo from Organic Disease of the Central Nervous System.**—This may accompany disease of any character situated in any portion of the brain; but it is especially liable to occur as the result of tumors, especially when they are situated in or near the cerebellum. Still it is perfectly possible, indeed, not infrequent, for tumors and lesions of the cerebellum to occur without the slightest disturbance of equilibrium. Vertigo is not an infrequent accompaniment of locomotor ataxia and disseminated sclerosis of the brain and spinal cord.

(*b*) **Vertigo from Cardiac Disease.**—This is usually the result of failing circulation. It is therefore aggravated by exertion, strong emotions, and causes generally calling upon the heart for extra work. It is especially a symptom of fatty degeneration of the heart. It is ofttimes the direct result of venous congestion of the brain. To trace a given case of vertigo to a cardiac cause is not always an easy matter, for the reason that in these cases there is frequently associated evidence of arterio-sclerosis and interstitial nephritis,—themselves capable, when acting alone, of inducing disturbance of equilibrium. Uncomplicated aortic regurgitation is frequently attended by vertigo, an act readily explainable by the constantly-varying intravascular pressure in that lesion. The vertigo associated with aneurysm of the thoracic aorta is due to the resulting disturbance of the cerebral circulation.

(*c*) **Epileptic Vertigo.**—Attacks of epilepsy (*petit mal*) are sometimes designated as vertiginous seizures by the patient. In such cases the attack comes without preceding warning, as is characteristic of epileptic paroxysms generally. Like Menière's disease, it may be associated with deafness and tinnitus. Epileptic vertigo, however, is readily differentiated from Menière's disease by the very quick recovery of the patient and the invariable association of some loss of consciousness, and the absence of any aural disturbance during the interparoxysmal periods.

(*d*) **Hysterical vertigo** is recognized by its remarkable features, unexplainable on any other hypothesis. The word-picture of the attacks, as explained by the patient, is greatly in excess of the conception of the cases, as understood by the examiner. The hysterical temperament and the usual stigmata of the disease are commonly present.

(*e*) **Neurasthenic vertigo** is dependent upon the readiness with which the hyperæsthetic nervous system responds to slight peripheral irritation. It is sometimes due to cerebral anæmia (?). The patient usually complains of such associated symptoms as general weakness, and sensations of pressure in various portions of the head.

Some of the cases of neurasthenic vertigo are really lithæmic, *i.e.*, the symptoms are associated with evidences of hepatic inadequacy. (See chapter on The Liver.)

(*f*) **Disease of the Special Senses.**—Common among the causes of vertigo is eye-strain, the special ocular defect being disturbance in the function of the eye-muscles. Vertigo is always a prominent feature of ocular palsies. It does not seem to be caused by the diplopia, for it still persists when the normal eye is closed. It appears to be due to the erroneous projection of the visual field. One quite often experiences vertiginous sensations when visual perceptions are at variance with perceptions obtained simultaneously through other senses. Cases of the above character are usually diagnosed without difficulty.

It is different, however, when vertigo accompanies simple eye-strain from refractive errors or muscular insufficiencies. Practically always we have, as suggesting the eyes as the primary cause of the trouble, such symptoms as blurring of vision, frontal and occipital headache, conjunctival irritation, and lachrymation,—all of these phenomena being aggravated by use of the eyes. The diagnosis, however, is based upon an examination of the refraction of the eyes by trial lenses, and the discovery of muscular insufficiencies by the Maddox prism or the phorometer.

(*g*) **Peripheral Vertigo.**—Two causes of peripheral vertigo have obtained special prominence. These are the laryngeal and the gastric. Laryngeal vertigo was first described by Charcot, in 1876. It begins with a sensation in the larynx which leads to a severe spasmodic cough, at once followed by vertigo and unconsciousness. The attacks last for but a few minutes. Physical examination shows quite a variety of conditions present in these cases. Almost any anatomical condition may be present; and, again, the larynx may present all the evidences of perfect health. By many, this variety is regarded as of epileptic origin.

Gastric vertigo, or rather vertigo attending disorders of the stomach, is probably the most frequently observed of all vertigoes. It is especially liable to attend acute and chronic gastritis and hyperchlorhydria. The most common cases are those succeeding too free indulgence in indigestible substances a short time before. Associated symptoms include foul taste, heavily-coated tongue, constipation, frontal headache, mental confusion, lassitude, and vomiting or nausea.

(*h*) **Toxæmia.**—The toxæmic conditions which produce vertigo are varied. Quite a number of poisonous drugs may produce it, *e.g.*, belladonna, cannabis indica, alcohol, tea, etc. Lithæmia, so-called, may give rise to very severe vertigo. The uræmic manifestations of Bright's disease include vertigo. In many of the cases of vertigo attendant upon interstitial nephritis this symptom is due to the associated high arterial tension, the arterio-sclerosis, and the cardiac hypertrophy.

(*i*) **Gerlier's Disease; Paralyzing Vertigo.**—This disease was first described by Gerlier; hence its name. It exists only within a very limited locality in Switzerland and Southern France. Its characteristic symptoms are vertigo, occipital pain, and certain palsies. These occur in paroxysms, which are usually of short duration,—that is, of not more than ten minutes. The paralyses are observed in the upper lid (ptosis), and in the extremities. The pathology of Gerlier's disease is obscure. By many it is attributed to a specific microbe; by others, the contagion of fear in a locality predisposed by superstition is regarded as an important factor. With the advent of cold weather the disease disappears.

(*j*) **Essential Vertigo.**—Nothing can be said of cases of this variety excepting that the vertigo seems to be the only symptom, and the cause thereof is undiscoverable.

5. **Aural Discharges; Otorrhœa.**—Ordinarily, aural discharges are derived from the external canal or the tympanic cavity.

They may be inflammatory or traumatic in origin. In the latter case the discharge may have been derived from the labyrinth.

A thin watery discharge may occur in the early stages of inflammation of the middle ear, indicating rupture of the membrana tympani and lesion of the labyrinth, caused by a fracture at the base of the skull.

A mucoid discharge almost invariably emanates from the drum cavity, and is due to inflammation of the same.

A thick, yellowish, purulent discharge may come from inflammation of either the auditory canal or the drum cavity. When from the drum cavity, its appearance in acute cases is succeeded by relief of pain. When from the canal, it is generally attended by diminution of pain; but the relief is not as prompt or as complete as in the case of middle-ear disease.

A bloody discharge results either from rupture of the membrana tympani or from granulation tissue, the latter in chronic cases. It is nearly always tympanic in origin.

A brownish, blackish or **bluish discharge** indicates bone disease.

Foul odor of a **discharge** indicates simply that there is decomposition from defective drainage. It is a common attendant upon bone disease, but has no necessary connection with that condition.

A discharge purulent in character, without preceding pain, is strongly suggestive of tubercular disease, especially if associated with multiple perforations of the membrana tympani.

If the discharge is of short duration, and recurs repeatedly, disease of the attic is to be suspected. Examination discovers perforations in the membrana flaccida.

Tests of the Auditory Function.—Sounds are conveyed to the brain by the ear or through the bones of the cranium. We test the acuity of the hearing by the watch and similar apparatus, or by the accoumeter of Politzer; by the voice, using the tone of ordinary conversation, the stage-whisper, or the reserve-air whisper.

The utility of the "reserve-air whisper" may be explained as follows: When one makes an attempt at whispering, he may utter the sounds with quite a range of intensity. Whispering of this character cannot, therefore, be regarded as a certain test of the acuity of hearing. But when, after a complete expiration, he makes an attempt at whispering, the sounds uttered have but one grade; in other words, they are a constant quantity. We may also use tuning-forks for the determination of pitch.

Before testing by the watch it is, of course, understood that the instrument is standardized by testing a number of normal cases. The record of the observations made is by a fraction, the denominator of which is the distance at which the watch may be heard by the normal ear, and the numerator the distance heard by the ear under examination. For example, if the watch is heard normally at thirty inches, and by the one under examination at twenty inches, then the fraction to be used in our record is $\frac{20}{30}$, or two-thirds of the normal hearing. When testing by the watch, it is important always that the ear opposite the one under examination be closed. To avoid any deception by the patient, it is likewise necessary that the eyes be closed. When practising the test, the operator should first place the watch at a distance beyond that at which it can be heard, and slowly approach it to the ear under examination. This is done in order to avoid the possibility of the patient being deceived by carrying the sound after once having heard it. In the use of the voice, vowel and consonant sounds are employed in the same way. The patient is directed to close his eyes, while the examiner, uttering a variety of words in a conversational tone, or with the " reserve-air whisper," approaches the patient gradually. The record of the result should be made : " Reserve-air whisper at such and such a distance ;" " voice at such and such a distance."

Although, in testing appreciation of pitch, tuning-forks of various pitch are used, the ordinary " C-fork " is usually quite sufficient, as forks are employed almost exclusively in the determination of the condition of bone-conduction. For this, two tests are commonly used, namely, those of Weber and Rinne.

Weber's Test.—The vibrating " C-fork " is placed with its base on the vertex, forehead, or against the teeth of the patient, who is requested to state in which ear it is heard the better. In the case of inflammation or disease of the conducting apparatus, *i.e.*, the canal or tympanum, it is heard more distinctly by the affected ear. If the trouble is due to labyrinthine involvement, it is heard better by the well ear.

Rinne's Test.—This is as follows : The vibrating tuning-fork is held with its base against the mastoid process. When the perception of sound ceases, the tines of the fork are held in front of the auditory meatus, and it will be noted that the sound is once more perceived. This corresponds to the normal condition. If, however, hearing is defective, such a state of affairs indicates labyrinthine involvement. In our record we note this result : " Rinne's test, positive." If, on the other hand, the vibrations are heard longer over the mastoid than with the fork in front of the ear, conduction by the bone is exaggerated, and shows involvement of the conducting apparatus. The term " negative " is to be used in designating the result in our record. In other words, when bone-conduction is relatively plus, we consider that there is involvement of the conducting apparatus ; when it is relatively minus, that there is a lesion of the auditory-nerve apparatus.

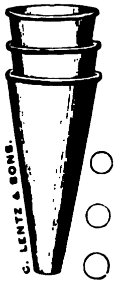
Neither of these tests is universally satisfactory. Numerous cases have been reported in which the inaccuracies of conclusions based upon them are evident. Under such circumstances, a correct diagnosis is to be obtained only after the study of all other associated signs and symptoms.

Objective Examination.—This is carried on with the aid of certain apparatus, as follows:

1. Head-mirror of six or eight inches focus.
2. Ear-specula, which may be made of various materials, as hard rubber, metal, etc.
3. Siegle's pneumatic speculum.
4. Eustachian catheter.
5. Auscultation tube.
6. Politzer bag.
7. Probe.
8. Cotton-carrier.
9. Pair of angular forceps.
10. Blunt ring-curette.

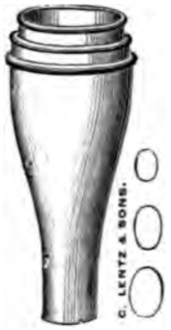
1. **The Head-Mirror.**—The head-mirror and source of light require no explanation additional to that given in the chapters devoted to the consideration of the nasal cavities and larynx.

FIG. 230.



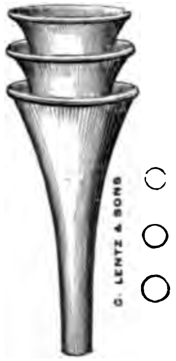
Nest of Wilde's ear-specula, with cross-sections showing different sizes.

FIG. 231.



Nest of Gruber's ear-specula, with cross-sections showing different sizes.

FIG. 232.



Nest of Toynbee's ear-specula, with cross-sections showing different sizes.

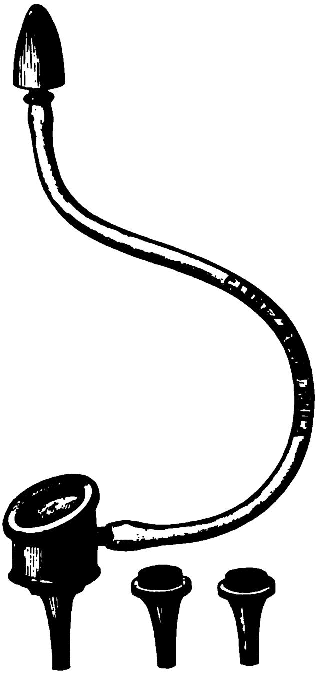
2. **Ear-Specula.**—Of these, several varieties in shape have been recommended, including those devised by Wilde, Toynbee, and Gruber. The latter are made of hard rubber or sterling silver, and are the most popular among otologists. They require no explanation additional to that afforded by the above figures and the appended legends.

3. **Siegle's Pneumatic Speculum.**—This consists of a hard-rubber speculum, the outer extremity of which is closed by a glass window, while the inner end is fitted with a rubber ring, in order to give it an air-tight contact with the bottom of the external auditory canal. The side of the speculum is fitted with a nipple, to which is attached a piece of rubber tubing by which the air contained in the instrument may be rarefied by suction by a rubber ball or the mouth of the operator. The latter is pref-

erable, because the rarefaction of air can be regulated better in this way than in any other.

4. **The Eustachian catheter** may be made of hard rubber, German or sterling silver. The latter is the preferable material, though decidedly more expensive. The annexed cut (Fig. 234) is self-explanatory as to the shape of the instrument. It is important that the operator have several sizes. The greatest care should be exercised in the cleansing of this instrument, for negligence of this precaution may readily carry infection.

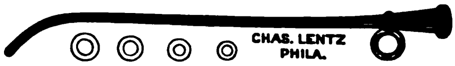
FIG. 233.



Siegle's pneumatic speculum.

5. **The auscultation tube** consists of a long tube of soft rubber, with ear-pieces, one black and the other white, to be inserted into the ears of the patient and operator respectively. It is more æsthetic, however, to dispense with the black ear-piece for the patient, inserting into his meatus the plain rubber, the end of which may be cut off immediately after using.

FIG. 234.



Eustachian catheter, with cross-sections showing different sizes.

6. **The Politzer bag** is to be employed for inflating the tympanum. The nozzle of the instrument is to be inserted into one of the nostrils, the

edge of which is compressed around it, the other nostril also being closed. The patient is instructed to blow forcibly through the mouth, and at the same time the operator vigorously compresses the bag, thus driving the contained air through the Eustachian tubes and into the tympanic cavities. (Fig. 240.)

Instead of directing the patient to blow forcibly, other devices may be employed. Thus he may be told to say "hic, hac, hoc, huc;" or he may make a forcible inspiration through the mouth; or he may take a mouthful of water, and at the moment of swallowing the same, the bag is forcibly compressed.

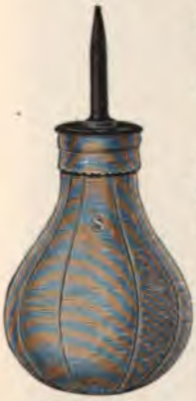
FIG. 235.

Auscultation tube.

7. **The probe** should be made of flexible metal, and its tip, before using, covered with the faintest layer of absorbent cotton.

8. **The cotton-carrier** should be of light construction, and is a necessary instrument in all cases in which the view of the parts is obscured by discharges. The many occasions in which a correct diagnosis is not made owing to imperfect cleansing of the canal is astonishingly large.

FIG. 236.



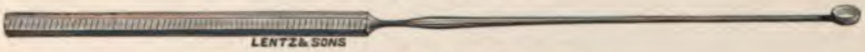
Politzer bag.

9. **The angular forceps** must be of the cross-branched variety.

10. **The blunt ring-curette** is to be used for the removal of scales of epithelium, particles of cerumen, and other solid materials obscuring the view of the membrana tympani.

Technique of the Examination.—Inasmuch as the external auditory canal has a curve with its concavity forward, it is essential, in order to straighten it and thereby gain a better view of its depths, that the auricle be pulled upward and backward; the speculum is then inserted, and may be held in position by the thumb and forefinger of the same hand which is making traction upward and backward on the auricle. (See Fig. 238.)

FIG. 237.



Blunt ring-curette.

In children, the curve of the canal is just the opposite to that found in adults. In them, traction should be made downward.

The main obstacle to inspection of the lower end of the canal lies in the projection of the anterior lower wall. There may be other obstacles arising from the presence of flakes of cerumen and epithelium, which must be removed with the probe.

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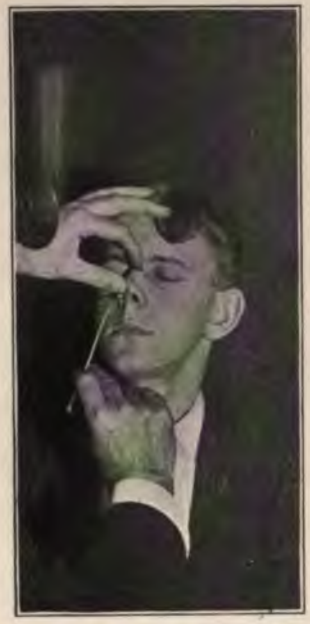
The speculum should be introduced as far as possible into the cartilaginous portion of the canal. Under no circumstances should it be pushed into the osseous portion, as the latter is very sensitive to pressure.

Normally, the auditory canal presents a white color. Small hairs or vibrissæ are numerous about its outer third. Small quantities of wax or cerumen and epithelial scales will also be visible.

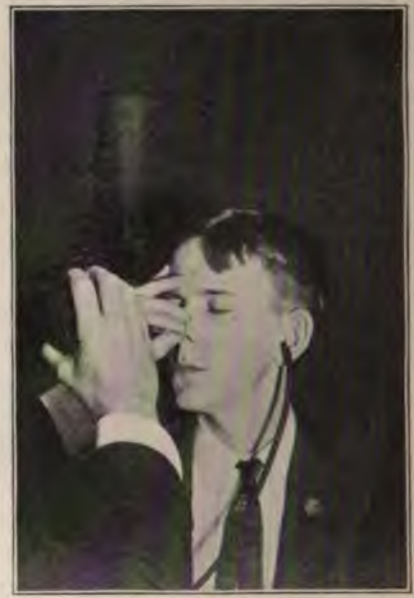
Throughout the examination, we must bear in mind the possibility of reflexes arising from the presence of the speculum, including especially the "ear-cough" and fainting. Very exceptionally, syncope, and even convulsions of epileptiform type, may ensue.

FIG. 238.



Method of inserting the Eustachian catheter.

FIG. 239.



Method of performing inflation by the Politzer bag and catheter.

The abnormal conditions of the canal include:

- Encroachment upon its lumen by the localized swelling of furuncle in its outer portion.
- A general narrowing of the canal from chronic diffuse inflammation.
- A nodular encroachment in the deeper portion dependent upon bone tumors, as hyperostoses and exostoses.

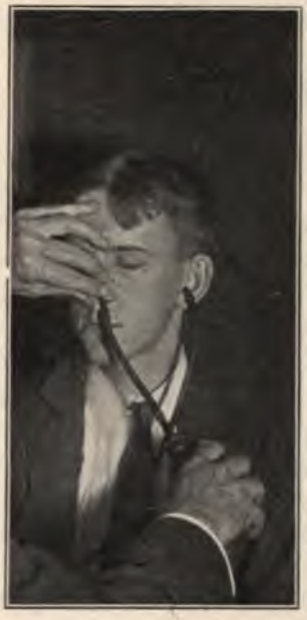
The lumen of the canal may be occluded by

- Scaly deposits of epithelium.
- Vegetable fungi (otomycosis aspergillus).

Discharges, which may result from
- Diffuse otitis externa.
- Furunculosis.
- Otitis media.

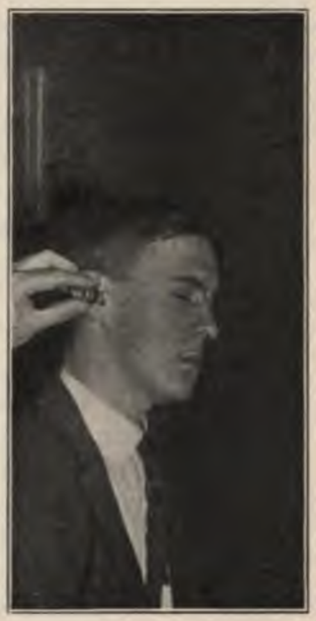
The Tympanic Membrane.—The membrana tympani lies at the bottom of the auditory canal, appearing as a translucent, polished, bluish-gray surface running from above downward, forward and inward, so that the anterior-inferior edge is further removed from the eye than the posterior-superior. A grayish line is seen running from above downward and backward, and represents the line of the hammer-handle. At the upper end of this there is a sharp projection, marking the position of the short process of the hammer. From this, two grayish lines project backwards and forwards, and are known as the posterior and anterior folds, above which the membrane is known as Shrapnell's membrane, or the *membrana flaccida*. The membrane below is known as the *membrana vibrans*.

FIG. 240.



Method of performing Politzer inflation.

FIG. 241.



Method of introducing the ear speculum.

The drum-head presents a slight, normal depression, greatest at about the centre, this point of maximum depression being known as the *umbo*, and it lies at the lower end of the hammer-handle. From this, running downward and forward, is a shining or reflecting triangular surface, with its apex at the umbo, and known as the light-reflex or cone of light. This varies in shape, in health, from a distinct triangle to a mere spot of light. In certain membranes, the long leg of the anvil shows through from the tympanic side.

The mobility of the membrana tympani can be made out most satisfactorily by means of Siegle's pneumatic specula.

The Eustachian tube may be examined by means of posterior rhinoscopy, although but little of practical value is to be gained by such a procedure. Its patency, together with the degree of ventilation of the tympanum, may be determined by three different methods, known, respectively, as the Valsalvian, the Politzer, and the catheter. By all three of these, air may be passed through the Eustachian tube into the tympanum. The sound made by the passage of the air may be heard by the examiner by means of the otoscope connecting the ear under examination with one ear of the operator. By this procedure we may determine by the character of the sound whether the tube is freely open or atresic, and the presence of fluid in the tube or tympanic cavity.

Abnormalities of the Membrana Tympani.—The polish of the membrana tympani is lost through inflammation, which may be of the external canal, or tympanum, through deposits of foreign materials, more particularly oils and medicines poured into the ear, or the presence of desquamating epithelium or wax.

Its translucency may be lost through inflammatory thickening, which, in turn, may be due to disease of the canal or of the tympanum.

The loss of translucency may be diffused or circumscribed. The most commonly observed of the localized opacities are calcareous spots, which usually indicate previous inflammation of the drum or old healed perforations.

The membrane may present a bright red surface, later gray and sodden, in acute otitis media.

Simple hyperæmia is indicated by the appearance of fine vessels running along the line of the hammer-handle and about its short process.

Exaggerated retraction is found in all conditions, attended by imperfect ventilation of the tympanum, as by closure of the Eustachian tube.

Retraction is indicated not only by the increased concavity of the membrane, but by exaggerated projection of the anterior and posterior folds of the short and long process of the hammer, which latter seem to stand out, at times, almost like a free skeleton of bone.

Flattening or bulging of the membrane will usually indicate the presence of free fluid in the tympanic cavity.

The surface of the membrane may show irregularities from scars, vesicles, polypi, and granulations.

Perforations of the membrane indicate previous inflammation of the tympanic cavity (probably suppurative in character) or traumatism. They may be single or multiple.

Perforations are to be diagnosed by inspection, or by means of air forced into the tympanum by the Valsalvian, Politzer, or catheter inflation. These failing in the presence of atresia of the Eustachian tube, we may help ourselves with the Siegle pneumatic speculum.

Pulsating fluid in the plane of the membrana tympani is usually pathognomonic of perforation, even when the perforation is not discoverable by inspection.

Polypi may be found growing from the cavity of the tympanum, or from the edge of a perforation, in chronic suppurative cases.

The ossicles may be entirely absent in chronic suppurative disease. The anvil is the most frequently lost; and the stapes the least frequently.

A Brief Synopsis of the Symptomatology of the More Common Diseases of the Ear.

1. **Inspissated Cerumen in the Auditory Canal.**—This condition arises usually from diseases of the glands, associated with pharyngeal catarrh, or from obstruction in the canal. The patient experiences sudden impairment of hearing, accompanied, at times, by pain and vertigo. There may be an irritating, tickling cough. Very exceptionally, most distressing symptoms may result from impacted cerumen, so that brain tumor and epilepsy have been simulated.

Objectively, the physician notes a blackish or brownish mass obscuring his view of the membrana tympani.

2. **Foreign Bodies in the Canal.**—These may consist of almost anything small enough to find entrance through the external auditory meatus. As to the outcome of this accident, it is not at all to be compared in its seriousness with that of the entrance of foreign bodies in the eye. In all cases, it is of the highest importance that the examination be conducted with the mirror and speculum, to make absolutely sure of the presence of the suspected body before acting. Most disastrous results have followed the attempts made to remove a foreign body which was never present. Impaction of foreign bodies in the auditory canal is always the result of improper instrumentation. The symptoms will naturally be those of irritation, including pain, sense of fullness, itching, etc.

3. **Diffuse otitis externa** is usually the result of the introduction of irritants, as rancid oils, etc., or other foreign substances. The subjective

symptoms include pain, soreness, itching, etc. Objectively, the canal is hyperæmic, and its calibre is narrowed by the swelling of its walls. Later, there appears a discharge of a thin membrane-like material and macerated cuticle.

Chronic diffuse otitis externa presents a thickened excoriated integument, with more or less narrowing of the canal, obscuring the view of the drum-head, and often an offensive flocculent or thick discharge.

Eczematous inflammation presents very much the same appearance as chronic diffuse otitis externa.

The appearance of scales or flaky membranes with black specks or dots and a thin discharge will indicate **otomycosis aspergillus,**—a parasitic growth, which, however, can be diagnosed with certainty only by the aid of a microscope.

4. **Furunculosis.**—Furuncles in the auditory canal may result from debility, anæmia, or over-feeding, plus infection and invasion of micro-organisms. The objective appearance of the lesion is that of a circumscribed swelling within the meatus. The symptoms include severe pain, which is worse at night and aggravated by any manipulation within the canal, and deafness. Furuncles involving the anterior or posterior wall are, as a rule, associated with sensitiveness on pressure over the parts anterior and posterior to the pinna. On this account, mastoid disease is sometimes mistaken for furunculosis. At times there may be œdema in front of the tragus or over the mastoid, thereby affording an additional reason for mistaking this condition for mastoiditis.

5. **Hyperostoses; Exostoses.**—These appear as circumscribed swellings in the deep portions of the canal. They are to be diagnosed by the absence of inflammatory symptoms, and touching them with a probe shows them to be very hard.

6. **Eustachian Catarrh** is usually the result of "colds," or of the exanthemata. Its symptoms are produced by the engorgement of the tubes, and the ensuing defective ventilation of the tympanum. They include sense of fullness, itching, pains in the throat, and cough. Among the ear symptoms are stuffy feeling in the ears, deafness, vertigo, and pain. The latter is sometimes extremely sharp. An examination with the speculum shows marked retraction of the drum-head. The condition lasts but a short time, and is promptly relieved by inflation of the tympanum, or it may go on to inflammation of the drum cavity.

7. **Chronic catarrhal otitis media** furnishes a large proportion of all cases of deafness. It is generally due to repeated attacks of nasal, pharyngeal and tubal catarrh. As predisposing causes of the latter we have post-nasal adenoids, enlarged tonsils, and obstructed nares. Gout and rheumatism also favor the disease. The symptom which usually drives the patient to the physician is a gradually-increasing deafness, usu-

ally associated with and sometimes preceded by tinnitus, and occasionally vertigo. In this deafness the appreciation of the lower-tone limit is impaired, and the tones of higher pitch are better heard. Bone conduction is exaggerated. Weber's test shows appreciation of the sound better in the bad ear. Rinne's test is "negative." Paracusis Willisii is not uncommon. Rarely, the patient complains of some pain and itching.

Objectively, the canal may be dry, or it may contain an unusual amount of cerumen. The membrana tympani is opaque and presents a dull surface, or it may be dull and atrophic. The light reflex is shortened or absent. The hammer-handle and the short process stand out prominently. The anterior and posterior folds are exaggerated. The drum-head is commonly impaired in its mobility. The Eustachian tube is commonly less permeable than in health.

8. **Acute Catarrhal Otitis Media; Earache.**—Acute catarrhal otitis media includes among its causes the extension of inflammation along the Eustachian tube to the middle ear, the introduction of fluids into the tympanum, improper bathing, traumatism, excessive or improper nose-blowing, etc. The disease is of rapid onset. The prominent initial symptoms include sense of fullness in the ears, deafness, tinnitus, and pain. The latter is the most prominent symptom. It gradually increases in severity up to the time the membrane perforates, when it ceases. It is generally aggravated at night or when the patient is lying down. The perforation of the drum usually takes place in from one to three days.

In children, the symptoms will sometimes simulate those of meningitis, because of the presence of vomiting, delirium, convulsions, and fever. The temperature in these cases may reach 103° to 104° F.

The attacks of acute catarrhal otitis media are at times so mild that a stain on the pillow on waking in the morning, with a dry deposit in the auditory canal, constitute the first evidences of the disease.

The objective examination of a case of catarrhal otitis media shows a diffuse hyperæmia of the drum-head, particularly of the lower part. With the stage of exudation there is flattening or bulging, and the surface of the membrane lacks lustre. After perforation, we note the presence of a mucoid discharge in the canal. In some cases perforation may not occur, the patient remaining deaf until the exudation in the tympanum is gradually absorbed.

9. **Acute suppurative otitis media** is a grave affection, the result of infection by one of the forms of pyogenic organisms in an acute catarrhal otitis or during the progress of one of the acute infectious diseases, notably scarlatina, diphtheria, and measles. Infection following traumatism of the drum-head is also a common cause. The symptoms are usually of sudden onset, and include great pain and elevation of temperature, accompanied by deafness, prostration, and often delirium. The

pain is not relieved until perforation of the membrana tympani takes place. The greatest hyperæmia of the drum-head is observed in its upper part. Sometimes the upper wall of the lower end of the canal is involved in the hyperæmia and swelling, so that it is difficult to make out where the canal ends and the membrane begins. In such cases, the operator finds it difficult to perform paracentesis, because he cannot tell into what structure he is cutting. A differentiation between the catarrhal and suppurative forms of acute otitis media can only be made by the rapidity of onset and the severity of the symptoms, and the prostration in the purulent variety. A maximum degree of swelling in Shrapnell's membrane, with involvement of the lower end of the canal, is, of course, indicative of the purulent variety. Perforation occurs quite frequently in the upper portion of the membrane.

10. **Chronic suppurative otitis media** is usually the result of neglected or improperly-treated acute cases, especially in scrofulous or tuberculous individuals. Very commonly, the only symptoms are a discharge from the ear—otorrhœa—and hardness of hearing. Many deaths result from this disease each year, by reason of pyogenic infection of the membranes and sinuses of the brain. By reason of improper drainage the discharge is often retained, and, undergoing decomposition, becomes very offensive. Exuberant granulation tissue and polypi are very common developments. The bones are apt to become diseased, especially in tubercular subjects. Those forms which involve the upper portion of the tympanum (attic suppuration) are usually the most serious. The obstructed drainage in these cases may cause fever, nausea, vertigo, and even extension of the inflammation to the mastoid, meninges, and the venous sinuses of the brain. Tinnitus is rarely complained of by patients with chronic purulent otitis. Perforation of the drum-head is always present, and may vary in size all the way from a barely-discernible pin-point to an open space so large as to leave a mere rim of membrane surrounding the lower end of the canal. The natural course of the disease is towards deeper and vital parts.

Complications of Purulent Ear Disease.—Of these, *mastoiditis* is the most common. As an idiopathic affection it is extremely rare. It may occasionally result from an acute suppurative condition outside of the drum, *e.g.*, multiple furuncles in the canal. The symptoms which first attract attention are extension or transference of the pain of the otitis to the region of the mastoid; tenderness on pressure over some portion of the mastoid, but particularly at the base, on a level of the meatus, or over the posterior portion of the apex. Of equal diagnostic importance, perhaps, is a redness and sagging of the upper posterior wall of the meatus, just in advance of the drum-head. There is usually elevation of temperature, though this may not be very marked or constant.

The swelling and redness over the mastoid, while usually present, is by no means a constant symptom. If present in the early stages, it may be regarded as a favorable sign—as indicating superficial or subperiosteal inflammation, rather than deep osseous inflammation; or it may indicate rapid advance towards the cutaneous surface, and away from the deep and more important structures.

11. **Disease of the Internal Ear.**—Knowledge of this subject is still meagre. Any of the suppurations of the drum may involve the labyrinth, especially in the course of measles and scarlatina. In the case of cerebro-spinal fever, the inflammation come from the side of the brain by way of the aqueductus vestibuli. Mumps and syphilis probably produce internal ear disease by way of the circulation.

In almost all cases of auditory-nerve disease, especially when the invasion has been sudden, the symptoms will be deafness, vertigo, due to invasion of the semicircular canals, and nausea. There are seldom fever and unconsciousness. The tuning-fork is heard better on the good side. The high register of sounds is impaired. Very sudden attacks are probably hæmorrhagic.

In 1861 Menière described a number of cases characterized by sudden invasions of paroxysms of tinnitus associated with vertigo, and followed by deafness. Sometimes the patient became partially unconscious during the seizures, and gave every evidence of profound circulatory disturbance. Since the discovery of this disease, literature has made the subject very confusing, because it is evident that authors describe very different pathological conditions under this name. The title "Menière's disease" should therefore be abolished, and, if the name of this physician is to be perpetuated in medical nomenclature, that of "Menière's complexus of symptoms" should be substituted. It is plain that these symptoms may arise in connection with any of the pathological conditions of the semicircular canals.

The symptoms of internal disease may have to be differentiated from those of epilepsy, brain disease, and bilious attacks. A study of the history of the case should guard against errors. The vertigo of labyrinthine disease is peculiar in typical cases, in that it is about a vertical or transverse axis. The patient very commonly falls in the direction of the affected ear. A good differential point to distinguish the vertiginous seizures from paroxysms of *petit mal* is the preservation of consciousness in the former.

12. **Presbyacusis, or senile deafness, is slowly progressive.** It is the result of sclerotic changes in both the tympanum and in the nervous apparatus. The impairment of hearing is apt to be greatest in the midst of noise. (See paracusis of Willis, or middle-ear deafness.) Usually the appreciation of high tones is impaired; cranial conduction is lessened. The membrana tympani may be either quite normal, thickened, or atrophic.

CHAPTER XXIII.

X-RAY DIAGNOSIS.*

THE discovery of the X-ray by Roentgen, in 1895, furnished both the medical and surgical diagnostician with an important addition to the methods employed for making known the nature of the disease or affection under inquiry. At first the surgeon profited most from the information elicited in this manner, but finally the medical practitioner also found the X-ray to be of considerable value in outlining certain organs of the body whose borders, by blending more or less with contiguous viscera, are uncertain of determination by the ordinary methods of diagnosis, such as percussion, auscultation, etc., but which become more or less defined by reason of the property possessed by the X-ray of making on the fluoroscopic screen shadows whose densities are practically in proportion to the densities of the tissues examined. Among the first of the organs of the body thus examined was the heart. In thin subjects this was found quite easy, and the diagnostician who possessed an X-ray apparatus was quick to appreciate the help it afforded in outlining this organ, especially when enlarged or displaced. The healthy lung was also found to be easily penetrated by the X-ray; but if the lung had fluid around it in the pleural sac, or was diseased in such manner that its density was increased, as in consolidation, or diminished, as in emphysema, these alterations were found to be made readily manifest in the fluoroscopic image on the screen.

Other organs of the body than those mentioned were soon added to the list of those capable of being examined by the X-ray, until every part of the body, even to the arteries and muscles, has been the subject, with more or less success, of X-ray investigation.

The surgical use of the X-ray is the one most generally known to the profession, though its employment by the medical diagnostician is becoming gradually more familiar.

This chapter will not enter into the question of the history of the X-ray, interesting and valuable though it is, it being ruled out by the nature of this work; nor can it discuss in more than general terms the apparatus employed. The diagnostician is assumed to be supplied with an outfit, and to know how to develop X-rays from it.

Outfits of various kinds and qualities are now numerous. The prospective purchaser will probably find it to his advantage to consult the users of X-ray apparatus, and, after a careful inquiry into the relative merits of the outfits they employ, he will be better able to put his money to good use. There are certain disadvantages in all X-ray outfits which time will doubtless overcome. Prolonged experience with any one of them, however, will finally enable the diagnostician to get the best possible service out of it.

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* This chapter has been prepared for the author by Dr. G. Maxwell Christine.

The writer has a twelve-inch coil of recent type, operated by a five-cell storage battery, having a capacity of ten volts for ten ampere hours. It has given general satisfaction, though a storage battery is at times necessarily a source of annoyance, owing to its uncertainty after use for a long time, and the need, if much work is done, for occasional recharging. This latter difficulty, however, can be obviated by connecting the battery with an Edison-light current, which will insure constant charging, or by having a second battery, fully charged, in reserve.

The Germans employ with success a Wehnelt break in connection with the coil, which does away with the condenser and the uncertainty attached to a storage battery. The tubes for use with such an outfit must be specially constructed, and are much more costly than the ordinary plain or adjustable tubes. Manufacturers have recently put on the market X-ray apparatus with rotary breaks, which give good service. Static machines have the disadvantage of being large and requiring a motor to run them. Those who use these machines, however, claim that their faults are outweighed by their numerous advantages.

Whatever outfit is used, care must, of course, be exercised to keep it in good repair, and to have it always ready for action. Nothing need be said here relative to tubes, except that it will be advisable to have on hand an assortment of hard, soft and medium tubes, unless the adjustable tube is used, when, as a rule, only one will be required.

The room in which the examination is made should be so arranged that it can be darkened at will. If the work to be done is simply skiagraphy, darkness is not so necessary ; but if it is fluoroscopy, relative and, at times, absolute exclusion of light is essential. Those who are expert fluoroscopists have comparatively little difficulty in the use of the instrument a few minutes after coming out of the light,—very much like the skilled ophthalmologist, who does not always require the use of a dark room to get a clear and distinct view of the inner eye. However, the beginner will best succeed if he remain in the dark room for eight or ten minutes, or wears dark glasses for that length of time, so that his retina will lose as much as possible of residual light sensation. Generally, while the tube is being put in operation and the patient is being adjusted, the eyes will get into the ordinary attitude of quiescence.

To many, the energized X-ray tube throws out enough illumination in the room to confuse observation. For this and other reasons the tube can be encased in a box lined with several coats of lead paint, and provided with diaphragms of various sizes to regulate the outflow of rays.

By use of this box, or some similar arrangement, the region of the body it is desired to examine will be the only part of the individual penetrated by the rays, which makes examination decidedly more satisfactory. A word of caution may be given respecting the use of a box to enclose the tube. The heat which is given off by the tube is often considerable, and it is sometimes a problem as to how best to conduct it away. If the box appears to cause inefficiency in the tube, the latter should be used without the box; or, the side may be open for more perfect heat radiation. A small electric fan may be set going toward the open end, in order to assist in carrying off the heat.

To some, the box is a nuisance, as they contend they get as good results without as with it. A simple substitute is to throw over the tube a black cloth, or to surround it with a paper box.

The patient under examination may be stood up before the apparatus, or seated in a chair having either a movable or a straight back. The better plan, in all cases where the organ to be examined is not affected by position, such as in pleural effusion, is to have the patient lie prone or supine upon some convenient bed, such as a canvas stretcher or trestles.

If the physician does not wish to be burdened with a special table or stretcher, a light invalid chair with back and bottom of canvas drawn taut, may be used, though, for general purposes, he will find the stretcher and trestles so satisfactory that their comparative increase in cost and the room they take up will be more than compensating. The energized tube is placed behind the patient, if seated or standing; and underneath, if lying down.

The X-ray aids in diagnosis by two methods, **radiography** and **fluoroscopy**. The former depends upon the action of the X-ray upon a photographer's plate, which enables the observer to preserve the evidence it affords for further study or use. The art has as its principle the action of the ray upon a sensitized film, the impression or picture thus made varying in degree with, in general terms, the density of the parts examined. Thus, if a piece of iron is placed over a plate and exposed to the X-ray, the shadow or picture produced will be sharply outlined; if it be a round bone, the density or thickness of the centre will cause the picture on the plate to be shadowed, as would be expected of an object having its middle portion thicker than its edges,—in other words, it is practically a "photograph taken through and through" the object. It is this property of the X-ray that enables the radiographer to take not only silhouettes of the organs he is examining, but to some extent a perspective view of them also. Nothing demonstrates this so beautifully as the picture of the hand of a young person in whom the epiphyses have not yet blended with the shafts. It is difficult to believe that the picture has not been taken by the ordinary methods of photography, rather than by those of the X-ray. To a more or less extent, this is true of any object whose parts vary in their index of X-ray penetration.

Fluoroscopy is the viewing of the parts under examination by means of the fluoroscopic screen or fluoroscope, the latter being a box-like adaptation of the former. Held up before the patient who is interposed between the energized X-ray tube and the fluoroscope or fluoroscopic screen, the screen is made fluorescent by the rays which pass through the individual under observation. This fluorescence varies in shadow according to the same rule that obtains in radiography ; but the latter is usually a finer demonstration, and shows effects not possible to obtain by the former.

Fluoroscopy may be said to be a view in gross ; radiography a view in detail.

For purposes of fluoroscopy, a good fluoroscope is essential. The best now made has a screen coated with barium-platinum-cyanide,—a device said to be due to the ingenuity of an American. It is essentially a box made to fit over the eyes so as to exclude all ordinary light, while the end opposite the face portion is occupied by a cardboard coated with the appropriate chemical. This device very quickly followed Roentgen's discovery, and has made the X-ray practicable for examination.

To view a patient with the fluoroscope, the following general instructions are all that it is necessary for us to discuss here. As before said, the room should be dark and the eye accustomed to the fluoroscope.

Moreover, the part to be examined should be kept in mind as to its normal condition, so that departures therefrom can be differentiated. The patient can be seated, stood up, or placed on the reclining-chair, or on a stretcher or table, as before described. The tube is properly energized, and the fluoroscope is taken in the hand of the examiner, his eyes peering into the open end of the box, the screen end being placed on the patient over the part to be examined. The portion of the tube giving off the greatest amount of fluorescence is directed toward the patient, whose distance from the tube, generally about two feet, will vary according to the activity of the tube. The part to be examined must be in a direct line with the tube ; and, as the X-ray travels in straight lines, in order to prevent distortion it is necessary to have the plane of the body at right angles to the direction of the rays. If the patient is sitting up, the tube will point its rays horizontally ; if he is leaning back, diagonally ; if lying down, then upward from below. With the details of position and relation of tube to patient attended to, the observer will look through the fluoroscope, and if fluorescence has been well established, shadows are observed in the fluoroscope. To the beginner these shadows may sometimes mean nothing, and may not even be perceived, unless very plainly manifest, as in viewing the hand. If the heart, the ribs or the spine are being examined, the novice may say he sees nothing definite, though to the experienced they appear distinct. This difficulty will, of course, disappear after a few trials. The fluoroscope may now be moved over the part, and as close to it as the box can be pressed without disturbing the patient, until all parts have been examined and studied. The energy of the tube may be altered to suit the desire of the operator; usually, a low vacuum will give a better fluoroscopic view than will a high vacuum. With the adjustable tube, adjustment is almost instantly secured by a change made in the length of the spark-gap of the auxiliary apparatus,—lessened to secure a low vacuum, or "soft tube," and lengthened for a high vacuum, or "hard tube."

The fluoroscopic screen is used slightly different from the fluoroscope. It is placed, as is the fluoroscopic box, against the patient,—opposite, of course, to the position of the tube,—so that shadows of the patient are pictured on it. It differs from the fluoroscope in the screen being open, and not boxed in. In some respects the screen is better adapted for examination than is the fluoroscope,—mainly when it is desired to get a large view of the parts and to make diagrams of the organs under examination, or to present the view to an audience.

If a fluoroscope of large enough size is employed, the screen portion, which is generally removable, can be separated from the box, and may do duty alone.

As at present devised, the use of the fluoroscope is limited to one observer at a time.

The patient should be stripped of all clothing likely to interfere with the finer details of the examination, such as corsets, buttons, buckles, etc., and enveloped in a thin garment. Nothing should be allowed to compress or in any way distort the part under examination.

The patient should be given an idea of the nature of the examination in advance, and an explanation be made of the steps of the process.

Otherwise, he may be startled or frightened, or, at an important stage in the examination, indulge in a movement of the body that will depreciate the value of the investigation.

A bandage thrown about the body or part under examination, and made to hold the patient to the chair, table, or part against or on which he rests, will often be suggestive of the necessity for quiet, and may save the operator the necessity for continually reminding the individual that he should not move. If the apparatus is apt at times to give off unusual noises, this possibility should be told him in advance, so that they will not surprise him into pulling away from supposed danger.

Thin patients are readily examined by the fluoroscope,—the ribs, the spine, the heart, the lungs and the limits of the diaphragm being very readily discerned. In the obese, fluoroscopy is sometimes difficult, and allowance must be made for the personal equation of the examiner's keenness of observation. This often amounts to considerable, because errors are liable in this method of diagnosis, unless all factors are taken into account.

The physician should spend some time in the examination of the healthy individual, first employing those subjects in whom the organs are normal as to size and situation. Taking the heart as the organ best studied, he should first determine its outlines by the usual methods of physical examination, making diagrams of what he finds. The patient can be seated, then allowed to lean back, and then to lie down, the varying alterations in the position of the heart, if any occur in these positions, being noted. After this, stouter individuals may be used as subjects, following which the physician is better able to determine such change in the organ as enlargement or displacement.

The same experience should be had respecting the lungs. If a case of large pulmonary cavity, as from tuberculosis, can be procured and compared with an emphysematous lung, the different effects produced on the fluoroscope will greatly aid in the study of differentiation. With one lung (say the right) normal, and the other having an accumulation of liquid in its pleural cavity, with consequent displacement of the heart, the difference in fluorescence of the two sides will be apparent. Other abnormalities as they present themselves can be profitably studied, of course in conjunction with the evidence furnished by other means.

It is a good plan to be supplied with skeleton sketches of the body, and fill them in with pen or pencil as soon as the examination is over.

In the use of the fluoroscopic screen the directions are nearly the same as for the fluoroscope, except that there is more need for an absolutely dark room with the former than with the latter. In fact, the more expert one becomes in the use of the fluoroscope the less he cares about the light in the room, provided he has a good apparatus in all its parts and his tube is potent. There must be, however, in the use of the screen, as much freedom from light in the room as possible, or the shadows on the screen will be dimmed and cannot be easily discerned. The screen may be fastened on an upright and held against the patient by an assistant, or even by the patient himself. If the latter is lying down, the screen may be placed over him, and bound there firmly by hand or bandage. On the screen may be placed a sheet of thin paper, on which the organs can be outlined with a pencil.

There is a device called a "*seehear*" with which auscultation can be performed while the observer is looking at the screen. Over this he may also draw his diagram.

Later, this will be more thoroughly explained.

One other very important use of the X-ray is **radiography,** already hinted at, to which a few paragraphs only can be devoted.

Radiography consists in the substitution of a photographer's plate for the fluoroscope or screen. The rays act upon the silver salts in the sensitized plate as do the rays of ordinary light,—not producing surface-pictures, as in ordinary photography, but shadows showing the various changes in the homogeneity of the part. The impression thus made on the developed plate is termed, as in photography, a negative, to which the prefix "X-ray" is added, making it an X-ray negative. When, by the ordinary processes of photography, this negative picture is transferred to paper, it is called a "radiograph," "radiogram," or "skiagram."

A few words only can be devoted to the method of making a radiograph of a part. The process differs very little from that employed in ordinary photography, except that a camera is not used.

In making a radiograph, the part is divested of as much clothing as possible, especially of all metal parts. If a limb is bound in splints, the latter need not be removed if they do not contain pins, etc. Even plaster-of-Paris casts are no bar to the penetration of the X-ray. To get good, quick results, however, the preferable plan is to have the part to be radiographed bare of all clothing, etc. The purpose is to have the rays projected from the energized tube pass around the part, and show it as a shadow on the sensitized plate. The *modus operandi* will vary according to the part of the body to be radiographed. If it is the hand or arm, foot or leg, the plate can be placed on a table or chair, and the hand or arm laid flat on it, with the tube about one or two feet above the plate.

For other parts of the body, it is best to use a table or reclining-chair. The purpose is to have the part at rest during the period of exposure. Most excellent work has been done by the simplest of methods, while elaborate apparatus has been used by some in the effort to get the very best results.

The sensitized plates generally used are especially made for the purpose, and are known as X-ray plates. They are usually furnished ready for use, enclosed in an inner dark envelope and an outer yellow envelope on which is printed blank spaces for making a record of the procedure, such as the name of the individual, the part radiographed, the time of exposure, etc. The outer envelope, after development of the plate, is generally used as a container. If much work of this kind is done, the plates are best purchased in their original box, and, about the time they are to be used, are placed in their respective envelopes in the dark room. X-ray plates should be used as soon as possible after their manufacture, and a statement should be always secured from the dealer as to when they were prepared. They should be kept in a dark, dry place, far removed from the influence of the X-ray. Some preserve them in a lead-lined box, but too much reliance should not be placed on its protective influence.

Before placing the plate in position the tube should be energized and put in proper condition for use. The patient is fixed in the desired position, and an explanation made as to what is expected of him and as to what he may expect. He should be particularly cautioned not to be startled and not to touch any of the apparatus. After the tube is found to be acting properly, the current to the tube is shut off, and the plate brought from its receptacle and placed in position immediately next to the part, but on the side opposite to the tube. The film side of the plate is to be next to the patient. If the plates are bought in their envelopes, the latter usually state which is the film side. If they are encased by the user, he should be careful to mark the film side "this side next to the patient." If desired, as a matter of safety, the encased plates can be put in a photographer's plate-holder, with the film side at the open portion. A bandage can be wound about the plates and the part to prevent displacement.

Everything being in readiness, the patient is cautioned to keep quiet, and the tube is again energized. The fluoroscope can be used to judge as to the efficiency of the tube, and, from the moment it is found to be acting as desired, the length of exposure should be timed by a watch.

At the expiration of the desired period, which varies from a second or two to several minutes, according to circumstances, the current is cut off and the plate is removed.

If fear is had as to causation of X-ray dermatitis, a medium of aluminum, or a wet cloth fastened in a frame and connected by insulated wire with the gas-main, may be interposed between the tube and the individual.

It is wise to do this at all times, as one can never be certain that he will not cause, in any particular case, a so-called X-ray burn. The public has learned considerable about this matter, and it is quite assuring to the patient to know that a means for protection is being adopted.

If desired, the plate can be developed while the patient waits for the result. If preferable, several exposures may be made, just as photographers do, the radiographer thus being reasonably certain that at least one out of the three or four negatives will be a success.

There is little opportunity in this article to give many details concerning the development of the plates in the dark room.

The writer uses metol-hydrochinon, which, with the proper directions, can be readily procured from the dealer. The plate is immersed in a weak solution for a few minutes to start development, after which the standard solution can be poured over it. Bromide of potassium can be added as a restrainer and to procure density. Unlike ordinary photographers' plates, the X-ray plate being of a double film, the impression does not show through the plate, and the process should not be pushed forward with the hope of seeing development on the reverse side. Usually, development can be stopped when the picture darkens out and all whites have disappeared. The developer is drained off and the plate washed under the spigot, and is then placed in a saturated solution of hyposulphite of soda to dissolve out the silver bromide. This usually takes about fifteen minutes, after which the plate is put in running water for an hour.

The plate is then swabbed off with wet absorbent cotton, after which it is stood up on the drying-rack. In a few hours the plate is dry, and, if the process has been successful, the negative will show a shadow-picture of the part radiographed.

Prints can be taken from the negative in the usual manner. Ordinarily, some such paper as Velox, which can be printed by gas or electric light, will serve all practical purposes, while at the same time it is most easily and quickly done.

In the examination of various unexposed parts of the body it has long been desired that, in addition to the ordinary means of diagnosis, there was some method by which, without exploratory operation, they could be exposed to view. It was this great need that caused Roentgen's discovery to produce such a startling commotion, particularly in the medical and surgical world. That the bones of the body could be outlined, that fractures, dislocations, disease of the bones, alterations in the position and size of the heart, consolidations and cavities in the lung, effusions in the pleural cavities, calculi in the kidneys, bladder and ureters, foreign bodies in the tissues, such as in the œsophagus, trachea, stomach, intestines, eye, etc., could be seen with the eyes, photographed on a plate, and reproduced in print,—that this could be done came like a flash to a profession which, since medicine started to be an art, had been waiting for just such a discovery. The most elaborate systems of diagnosis, setting forth as reliable methods of determining the nature of the disease under examination as science could furnish, have always been short of the one great essential to the proper estimate of internal disease-conditions,—the ability to see the part. This need for a means by which ordinary diagnostic measures could be supplanted by others which would assist the eye is well illustrated in a tumor in the region of a bone. The question arises, is it an enlargement of the bone itself with overlying soft tissues, or is it mainly composed of soft tissues, replacing bone structure which has been destroyed by a pathological process ? The differential diagnosis between a bone hyperplasia and a bone necrosis has always been a matter of uncertainty, largely because of the obvious difficulty of determining whether the enlargement is bone-overgrowth or necrotic or malignant tissue. The X-ray came as a welcome aid to the surgeon, because it enabled him to see through the tissues and thus determine definitely their nature,—ability to do which he did not possess prior to Roentgen's discovery.

Diagnostic methods have frequently to be discounted because of their uncertainty, and each year we find a new one being added to the many we already have at our command. Those who have practiced medicine a quarter of a century, for example, have witnessed marvellous changes in diagnostic methods ; and though, superficially, it would seem that they cannot be improved upon, yet when we carefully study the matter, and come face to face with the fact that often there is uncertainty attached to a diagnosis, no matter how carefully and scientifically worked out, we are forced to the conclusion that other methods are still in store for the diagnostician, and that this uncertainty to which we have referred can be lessened, and finally be replaced by unquestioned certainty. The surgeon who found that he could add the X-ray to his usual means of diagnosis of a bone tumor jumped at once from uncertainty to reasonable certainty, and the great need which he had felt for a more scientific determination was most satisfactorily met.

Twenty-five years ago pus in the urine was, as to cause, conjectural in the majority of cases. The elaboration of the means by which that cause could be determined has been most interesting, and the various means of exploration of the urethra, bladder, ureters and kidney, made possible by the invention of various devices for the purpose, placed genito-urinary diagnosis and surgery on a very high plane ; but with all the precision which this advancement in diagnostic skill made possible, something was wanting—the ability to see the part diseased. Thus, as a result of this great need, came the endoscope, the urethroscope, the cystoscope, ureteral catheters, etc. The principal cause of pus in the kidney, of obstruction in the ureter, and often of cystitis, is stone in one or other of these organs. Years of careful study have been devoted to perfecting our ability to determine beyond doubt not only the existence of stone, but its relative position. No known means, except exploratory incision, gave the diagnostician the positive information he desired, until the X-ray came to his assistance ; and, while even this has its limitations, those who were specially skilled in this line of diagnosis promptly resorted to the use of the X-ray, —in some instances for its positive, in others for its negative, and in still others for its confirmative, information. Stone in any one of the three mentioned organs may be diagnosed by the ordinary methods, but there yet may be a doubt which will interfere with the institution of appropriate treatment. The X-ray has caused this doubt to be measurably removed by enabling the diagnostician to supplement his usual procedures with one of extraordinary confirmative value. Thus, in the diagnosis and treatment of renal and other calculi, to perfect which many years of careful and systematic research had been devoted, and which, prior to the application of the X-ray to this branch of the medical art, was thought to be as near perfect as it was possible to make it, a great stride was suddenly taken by the ability of the examiner to photograph an opacity in a region which, by other methods of diagnosis, was assumed to be a stone. Hence every one wishing to perfect himself in either the medical or surgical treatment of genito-urinary surgery resorts to the X-ray for the diagnostic information it gives. To the circumstantial evidence as afforded by urinalysis and by the symptoms, etc., the X-ray enables us to *see* and to *picture* the organ under investigation, and, by comparing its appearance when abnormal with its appearance when normal, we are in a position to add the evidence of our visual powers to the evidence obtained by other means.

Another illustration is the presence of a needle in the tissues. Examination frequently offers no evidence whatever that there is a needle in the flesh, and yet a needle was known to penetrate the tissues and become lodged there. Surgeons know how difficult it is to ascertain the presence and the exact location of the needle so embedded. Tissues have been viciously incised and explored in the vain endeavor to find the needle. When the X-ray came to the help of the surgical diagnostician, how easy it became to photograph the opacity in the tissues, and thus remove the uncertainty attached to its presence and location?

These are surgical illustrations largely, but they illustrate the great diagnostic importance of the X-ray, and assist us in emphasizing the fact that, however valuable other means of diagnosis of internal conditions may be, any additional means of diagnosis which will expose the part to our view is of stupendous value.

And yet, in estimating the X-ray as a diagnostic aid, we must not become so enthusiastic as not to understand its limitations. The surprising ease with which the internal portions of the body may be viewed induced us, in the beginning of our application of the Roentgen's ray to diagnosis, to entertain extravagant notions as to the evidence it furnished. For instance, the fluoroscope is used on the hand or arm of an individual, and we announce that we see something which is narrow and about an inch long. This is an opacity simply. We may say, if we analyze it, that it looks like a pin, or a needle, or a piece of wire. Then, again, we may say that the opacity appears to be in the hand. Of this we are not certain. It may be in the hand nearest the fluoroscope or farthest away from it. It may be outside the hand, and not touch it in any of its parts. We may say it is in the tissues of the hand, but the fluoroscope, as thus held in the one position against the hand, affords no such evidence. Neither does it indicate more than circumstantially the nature of the opacity. Before any value can be attached to the fluoroscopic or radiographic image, certain points in the evidence are required. In other words, it needs interpretation, and much evidence that is collateral is required to make this intelligible. In determining where this opacity is, a knowledge of all the facts bearing on the case should precede the examination. A certain pathologist of Europe always requires a full and complete history of every pathologic specimen to be given him before he will attempt a diagnosis of a slide offered him for examination. He insists on knowing especially the organ and the part of the organ from which the specimen was taken. So, in determining the nature of the little opacity we see in looking at the hand with the fluoroscope, we must know, first, that it is a hand we are examining; second, that there are no foreign bodies on the surface, or near it, or anywhere near the fluoroscope, likely to give a false impression as to the existence of a body within the hand. We must be furnished with a history of the case; if it is an injury, then we must be given to know its general nature; and if a shot, or a needle, or some such body has entered the tissues, we must be informed as to the point of entrance and the probable direction in which it went through the tissues. With these and any other data the examiner may specially require, he is in a position to institute his investigation as to the presence or absence of a foreign body. Of course he must know the anatomy of the part he is examining, so that he will not confuse the normal with the abnormal. Examining the hand, he outlines its soft tissues and the bones, noting the joints and the sesamoid bones, if they are prominent enough to be seen. If the hand is normal he should be able to so determine, so that, if he sees an opacity, he is in a position, with all the necessary data before him, to say that it is within the hand. Then he is to determine, by varying his examination, the exact situation of the opacity; and, finally, he may sometimes, by a method of comparison, even determine reasonably the exact nature of the opacity.

We have devoted space to this matter because the X-ray simply deals with shadows made by opaque objects, and determines nothing more than their existence, size, form, and situation. This limits the X-ray, for purposes of diagnosis, within somewhat narrow confines; but, even at that, its value is inestimable.

Referring again to the needle in the tissues, let us suppose it has penetrated and is lodged in the knee. If the X-ray will determine for the surgeon the existence and exact location of a foreign opaque body resembling the missing needle, the surgeon cuts to the point indicated on the radiograph, and exposes to view the needle with the greatest possible minimum of destruction or severance of tissues. A piece of steel enters the eye, and in this particular instance the ophthalmoscope does not show its location, nor does any known means indicate positively even its existence within the eye. A radiograph shows, however, that there is an opacity similar to a piece of steel at a certain spot within the organ. The surgeon cuts down to the spot indicated on the diagram given him by the radiographer for his guidance, and the piece of steel is found.

The value which the X-ray is to the physician or surgeon is one simply of enabling him to observe, by means of the shadow-pictures they form on the fluoroscopic screen or photographer's plate, first, the various tissues and organs of the body which, by reason of their difference in density, etc., permit such differentiation, in this way enabling a study to be made of the normal and of the abnormal; and, second, the existence of bodies which may be accidentally or designedly within the tissues.

With this understanding of the fundamental principle of the X-ray in its application to medical and surgical diagnosis,—namely, first, that it simply outlines the contours of the body, secondly, that it outlines certain organs and tissues of the body, and, thirdly, that it outlines and locates foreign bodies which may be within the tissues, all of which is done by a differentiation of shadows,—we are in a position to take up the subject of the use of the X-ray in assisting the medical diagnostician in determining the nature of certain internal diseases.

This is to be done by comparing the shadow of the part under examination with the shadow of the part in its normal condition.

As this chapter excludes all reference to surgical diagnosis and is confined to clinical medicine, we will take up the various divisions of the body, and study in a general way the aid the X-ray is likely to afford in diagnosing pathologic conditions associated therewith.

The Cranium.—In an article in the *Philadelphia Medical Journal*, Sept. 27, 1902, Mills, Pfahler and Deaver present the history of a brain tumor which, after being clinically localized, was also located by the Roentgen ray. They state that this was the third brain tumor which, during the life of the patient, was shadowed by the X-ray, the second having been reported in the *Philadelphia Medical Journal* for February 8, 1902. In the *American Journal of Medical Sciences* for February, 1899, Church presented the history of perhaps the first case of a brain tumor so demonstrated by the X-ray after being clinically localized.

These demonstrations of the existence of abnormal shadows within the skull, which by clinical means were diagnosed as cerebral tumors, mark a marvellous advancement in the direction of the application of the rays to the diagnosis of brain lesions. Williams, in his admirable work on the X-ray (1901), states that a tumor in the brain must necessarily offer a good deal of difficulty as to detection by the X-ray. This is obviously true because of the inherent difficulties attendant on the passage of the rays through the skull, and the ability to properly interpret the normal shadows as compared with others which may appear abnormal.

Before the examiner can hope to profit by the use of the X-ray in brain work he must be well versed in the normal shadows. Pfahler, in the article referred to, indicates quite clearly the normal shadows to which we must pay special attention. He states that before reliance can be placed on any abnormal shadows in the skull or brain it is necessary to obtain certain details as to the scalp, the outer and inner tables of the skull, the occipito-parietal and fronto-parietal sutures, the frontal, ethmoidal and sphenoidal sinuses, the roofs of the orbits, the sella turcica, the external auditory meatus, and the mastoid cells. He accompanies the article with a fully-explained cut of a skiagraph of a skull with enclosed brain tumor, which can be referred to by the reader for profitable examination and study. The tumor in this case was the largest Mills has ever seen taken from the living human brain. It is probable that small tumors and other diseases of the brain will for some time offer considerable difficulty to the X-ray clinical diagnostician ; but as apparatus and methods become perfected, and our interpretations of the shadows of the skull and brain are made more intelligible, much fine work in this direction will doubtless be accomplished. Clots large enough to form shadows may some time in the future be diagnosed by the X-ray, but our present knowledge does not now permit it.

Spinal Column.—The X-ray has not yet proved of any service to the medical clinician in his diagnosis of spinal-cord affections. The shadows formed by the spine are too indefinite to warrant much being said of them. A tumor of the spinal cord would cast a shadow were it not blended-in with the complex bony structure enclosing it; but it is impossible to place any interpretation on any shadows it casts as indicative of cord changes.

The Thorax.—Made up of a bony encasement, with the spinal column in the centre posteriorly, the ribs extending therefrom around and forward, and uniting to the sternum in front, capped at the top with the clavicles and overlaid posteriorly with the scapulæ, the thorax contains certain organs which, by reason of their difference in fluorescent penetration, cast varying degrees of shadows when examined with the X-ray. The lungs, the heart, the aorta, the diaphragm, with the liver beneath, cast shadows which are pretty constant in the normal individual.

These need to be carefully studied in order to compare them with shadows which may be cast by such abnormalities as pulmonary consolidations, cavities, emphysema and tubercular disease, tumors in the mediastinum, aortic aneurysm, enlarged or displaced heart, pericardial effusions, pleural effusions and empyema, tumors of various kinds, foreign bodies, etc.

With the patient seated upright in a firm position, reclining in an easy-chair with canvas or light wood back, or lying on a suitable table or stretcher, the thorax can readily be examined for abnormal shadows. The clinical history will probably point toward the portion of the chest where there is a suspicion of an abnormal condition existing, which part should be most carefully examined, with the patient in all three of the upright, reclining and horizontal positions. With the mind of the examiner strongly bent on the normal, any existing abnormal shadow must be closely studied and carefully interpreted.

Supplementing the fluoroscopic view, radiographs on plates sufficiently large to include the whole of the thorax should be taken, first with the plate placed posterior, and, secondly, anterior, to the chest. The respiratory movements of the thorax can be allowed free latitude in using the fluoroscope, but in radiographing they must be restricted as much as possible, to prevent diffusion of shadow-outlines. If the tube is sufficiently energetic, the patient may be told to hold his breath for ten or fifteen seconds, during which period a good radiograph can often be taken. If this length of time is not sufficient, the patient may be directed to hold his breath on two or three separate occasions, during which time the rays should be allowed to pass through the plate, a thick leaden disc being placed under the tube during the breathing and removed during the spells of rest. This will give the requisite time-exposure with a minimum of chest-movement.

The normal lung permits the X-rays to pass through without much interference, so that it is easy to examine with the fluoroscope, and radiographs are also easily taken. The right lung from the apex to the diaphragm will have no shadows except the clavicle and the ribs, which in the ordinary individual will not be very marked. If the respiratory movements are not impeded, the thorax will enlarge with inspiration and diminish with expiration. As the fluoroscope rests on the chest, it will move with the chest-movements. But at the lower border of the lung there will be observed a shadow which markedly rises and falls with each respiratory movement. This is the shadow cast by the diaphragm, which it is important to carefully study in pulmonary X-ray work, because of its limited excursion in certain diseases, as in pulmonary tuberculosis,—a disease in which this limitation is most pronounced and becomes a diagnostic point of importance.

Williams gives particular attention to the X-ray diagnosis of tuberculosis of the lung, and lays great stress upon the limitation of the shadow cast by the diaphragm, which he says is restricted on the affected side or sides, and usually in the lower part of its excursion. The diseased lung is increased in density in proportion to the area of the tubercular infiltration, and hence the affected lung or lungs will be darker in shadow than the healthy portions. In all examinations of the lung, as indeed is also true of all organs, it is to be recollected that the degree of fluorescence will depend not only upon the activity of the energized tube, but also upon the thickness of the chest of the individual. Large amounts of fat over the chest-wall will also lessen the fluorescence, and must be allowed for. If one lung is affected and the other is sound, it is well to take the fluorescence of the sound lung as the index by which to compare that of the diseased lung ; or the base of one lung may be normal and the apex consolidated, in which case the lower portion of the lung is taken as the basis of comparison. Williams claims that the heart may be drawn toward the affected side, especially during deep inspiration. In disseminated tuberculosis of the lung it requires keen judgment to determine that the shadows cast by it are abnormal ; but experience will soon enable the examiner to differentiate these disease-conditions which to the novice may appear meaningless. Radiographs show anywhere from a mottled appearance of the lung to a deeply dark shadowing, the reason for which the nature of the disease makes plain.

The shadowing and restricted diaphragmatic excursion on the affected side of the thorax may not, however, always be indicative of tuberculosis. Recent pneumonia and pleurisy may do the same. In all cases the history of the disease must be associated with the X-ray diagnosis and interpretation. Those who desire to add X-ray diagnosis to their usual methods will become more and more convinced that it is not only confirmative, but is often suggestive and conclusive. Even when tuberculosis has not been suspected, X-ray examination of the lungs has demonstrated its presence.

Increased shadow in the lung may be due to pneumonia or congestion of the organ, or to pulmonary cavities filled with exudate.

In one case of the writer's a pint of pus was expectorated which had evidently broken into a bronchus from a pleural empyema, which had been mistaken for a pneumonic consolidation. Thoractomy was finally performed, and drainage maintained for some time by means of a rubber tube. During the treatment of this patient, prior to operation, the perfect fluorescence of the right lung with normal excursion of the diaphragm, displaced heart to the right, diminished fluorescence of the left lower thoracic cavity due to the accumulated pus,the nearly normal fluorescence of the apex, the difference in level of the shadow with the patient lying down and sitting up, were most admirably demonstrated by the X-ray, which proved itself to be a better means than any other now known of confirming clinical indications in diseases of this character. As Williams says, it is a most valuable method for controlling the results obained in other ways.

A shadow seen in the right thorax may be due, as shown by Waldo and quoted by Walsh, to cirrhosis of the lung, which, by its contraction, has drawn the heart over so far to the right as to cause the apex-beat to be outside of the right nipple. In this case the Roentgen ray showed an abnormal shadow in the right lung, thus confirming the clinical diagnosis.

If the diaphragm shadow is lower than normal, we may reasonably suspect emphysema of the lung, or pneumothorax.

If the usual lung-fluorescence is interrupted at any point by an oasis of intenser light, we may assume there is a cavity filled with air, which, of course, would need to be confirmed by other tests to make such a diagnosis positive.

In examining the shadow cast by a suspected pneumonic lung, consolidation shadow may be found under a portion of the chest-wall which responds normally to auscultation and percussion, the consolidation being too deep for detection by the usual signs. This is also often the case in tubercular lung affections, in which the physical signs are entirely absent.

As the congestion and consolidation of the lungs increase or diminish, the density of the shadow will be proportionately greater or less.

If the pleura is thickened, the shadow will be darker than when that membrane is free from inflammatory deposit. Usually we will find the pneumonia shadow to be in the middle portion of the lung, with the apex and base very slightly if at all shadowed. As the disease progresses, the shadow enlarges.

The shadow seen in the thorax of a pneumonia convalescent will usually grow less as the disease improves. If, however, as time goes on, the shadow grows darker at the base, we may suspect empyema of the pleura, as already indicated in the case previously cited.

In a suspected case of liver abscess in which dullness on percussion extends up into the lung area, the fluoroscope may show only slightly subnormal diaphragm excursion, while the darkened shadow at the base of the lung may not be very dense. These conditions would imply that the disease is not an abscess, but a consolidated lung at its base.

If, in examination of the chest, we find the fluorescence to be unusually bright, and extended in area farther upward at the apex and downward at the base than is normal, with diminished diaphragm excursion, we may diagnose emphysema of the lung.

The heart, which, by reason of the overlying emphysema, is often made out with difficulty by the ordinary methods of diagnosis, is easily demonstrated by the X-ray as a rhythmically-moving shadow.

If the emphysema interferes with the physical examination of other portions of the thoracic contents, such as in tuberculosis, cardiac enlargements and displacements, aneurysmal and other tumors, the X-ray will cast their shadows, and thus demonstrate their existence when other known means might not have done so. Of course, the mere existence of shadows under such circumstances will not always be sufficient to render a correct diagnosis possible, but they will point to the need of a careful study of their meaning. The fluoroscopic shadow always presupposes intelligent interpretation.

If a thoracic shadow is suspected to be the heart, it is necessary to exclude all internal thoracic diseases which may cast confusing shadows.

As already stated, all X-ray examinations should follow a careful history-taking and physical examination of the case; so that the examiner may, if possible, comprehend whether there is any condition likely to complicate the heart-shadow. Usually the pulsations of the heart will enable differentiation between that organ and any other body whose shadow blends with it. The shadow of the spine unites with that of the heart at both its upper and lower portions, and may confuse the beginner in X-ray work ; but after a few examinations this difficulty will largely disappear. The fluoroscopic vision will show a decided difference between the fixed column and the pulsating cardiac shadow. Inspiration and expiration will not affect the spinal or sternal shadow, but it will the heart. It has been well demonstrated that during a deep inspiration more lung- and heart-surface is brought into fluoroscopic view ; hence the heart is more easily seen. Moreover, during quiet or arrested breathing the pulsations of the heart are more plainly seen, which fact also affords a means of distinguishing between the heart and spine or sternal shadows.

Other shadows are those of the diaphragm, liver, and the large blood-vessels. The latter, when large, and particularly when dilated, pulsate, and may often be distinctly outlined in contrast with the heart. Shadows seen in the region of the heart may be due to tumors or enlarged glands, and to atheromatous arteries. It must be remembered that the fluoroscopic shadow of the heart will usually be larger than will be the outline made on the chest by the ordinary physical examination. This is due to the fact that some of the shelving border of the heart does not present itself against the chest-wall. The shadow is therefore greater than is the area of dullness.

If, in pleural effusions, in addition to the shadow cast by the liquid in the left chest, there is a pulsating shadow in the right chest, it is evidence that the heart has been displaced.

If, in the examination of a chest, the left side is lighted up brightly without the usual heart-shadow, and on search the heart-shadow is found on the right side, that organ has been transposed.

Some observers claim to have been able to distinguish the beating of the auricles in dilatation.

Thoracic aneurysms are usually readily discovered or demonstrated by the X-ray. Their shadow is a pulsating one unless the tumor is filled with a clot, in which case pulsations are impeded or absent. In a shadow supposed to be that of an aneurysm, vertebral, sternal and other shadows must be carefully excluded.

Differentiation is not always easy between thoracic aneurysm and intra-thoracic tumors. The pulsations are not always present in the former, and their absence may mislead. The most thorough use of the fluoroscopic screen in various positions, front, back, and at the sides of the chest, may serve to outline the enlargement, which physical examination may further explain. Sometimes a shadow will reveal a condition not previously known to exist. In a case with dyspnœa or any other of the symptoms of aneurysm occurring in a patient not suspected of having that disease, the screen may show an abnormal shadow, which may for the first time indicate the exact nature of the affection.

If the shadow is due to a growth, it may be found to be situated at a point not normally occupied by the large vessels, in which case differentiation is made easier. The growth may be next to a large blood-vessel, and thus receive pulsations which may also be misleading. If aneurysm is suspected but the fluoroscopic examination is negative, the presence of aneurysm may be reasonably doubted. Hence the absence of shadow becomes of equal importance with the value of its presence.

Not only in thoracic, but in subclavian and other aneurysms, the X-ray will be found to be of inestimable value as an aid to diagnosis.

In all cases of examination, the radiograph should supplement the investigation by the fluoroscope. In this way comparison may be made not only between the results of these two diagnostic methods, but between them and the evidences pointed out by physical examination.

Abnormal shadows in the neck, thorax and abdomen may be due to alterations in other organs of the body than those mentioned, such as the œsophagus, stomach, etc. The œsophagus and stomach are ordinarily difficult to outline. Mechanical aids within the canal and gastric pouch are usually required to give them outline. Distention by flexible sounds for the œsophagus and water for the stomach may afford the necessary means for outline. Subnitrate of bismuth will coat the stomach in such a way that it can be shadowed. The contents of the stomach and intestines should be cleared out by stomach-tube, purgation, etc., before the examination, after which the flexible sound or bismuth may be introduced. The stomach can be distended with gas, and by the increased area of fluoresence its outline may be judged. Dilatation, position and contraction may thus be diagnosed. Williams uses one ounce of pure bismuth well mixed with bread and milk, after which the examination is made.

The liver and spleen cast shadows, but it cannot be said that the X-ray is of any special value over and above the ordinary and physical examination. The upper border of the liver may readily be determined, and the outlines of the spleen may at times be differentiated from other organs, especially if the stomach and intestines are distended with gas.

Shadows in the renal region which make the X-ray of special value are those of renal calculi. Displaced kidneys, tumors, abscesses, etc., can also be detected by the X-ray. The clinical diagnosis of stone, whether in the kidney, ureter or bladder, is more or less constantly uncertain. It is in this class of cases that the X-ray has been of the most brilliant service.

If, in the region of the pelvis of either kidney, or along the route of one or the other or both ureters, or in that of the bladder, the X-ray shows a shadow of the usual size of a calculus, and especially if two or three examinations demonstrate in all of them a like condition, the inference is that the shadow is one of stone. It need not necessarily be so, however, for mistakes have happened in the use of the X-ray for this purpose. If the symptoms indicate the disease, and the clinical diagnosis fairly well establishes the existence of calculus, the X-ray will be an important aid in confirmation. In other instances it will clarify the clinical picture, and, by demonstrating an abnormal shadow in a position highly suggestive of stone, the clinician's inquiry will naturally be directed toward the determination of its true meaning.

The fluoroscope is useless in the determination of the presence of stone. The radiograph should be large enough to show definite relationship between the parts, such as the ribs above, the spine in the centre, the pelvis below, and the soft tissues to the outside. The tube is best about twenty inches from the plate, and over the third lumbar vertebra. The plate is best placed at the back, the tube being over the abdomen of the patient. Leonard has pointed out that this gives the clearest view of the pelvic outlet, the bladder, and the prostatic portion of the urethra.

Every radiographer recognizes the fact that possible defects in his plate, due to inherent faults in the film or development, etc., require the exercise of the greatest care in the work. Hence it is well, whether the examination is apparently clearly affirmative or negative, not to rest content with one negative, but to have at least three. If each confirms the others, the evidence is the stronger.

To illustrate the value of the X-ray in the diagnosis of calculi, Leonard states that in 227 patients 68 showed calculi, 43 of which were in the ureter. Forty-seven cases were in males: 18 in the kidney and 29 in the ureter. Twenty-one were in females: 8 in the kidney and 13 in the ureter. In 11 cases bilateral calculi were found, sometimes renal and ureteral. In 5 cases, multiple ureteral calculi were found.

As mentioned early in this chapter, the diagnosis of renal disease has undergone marvellous development; but the positive diagnosis was wanting until the X-ray came to take an actual picture of the stone in its resting-place deep in the tissues, where no human eye could see it or hand feel it except by incision. Exploratory incision is now unnecessary, and the operator for stone has it in his power to have indicated for him with practically unerring accuracy the exact spot for his operative procedure.

Biliary Calculi.—Shadows of bodies in the vicinity of the gall-bladder and ducts may be those of biliary calculi. The rule observed respecting renal calculi, in the matter of taking several radiographs, will be found to work well in radiographing for biliary calculi. Radiographs intended to include the gall-bladder and ducts are best taken with the patient lying on the plate, with the right side pressed firmly against it on the table, so that, as King points out, the rays will not penetrate the abdomen vertically, but from side to side, so that they form an angle of 45° with the plate.

Arthritic and gouty deposits may be shadowed by the X-ray, and differentiated from other diseases likely to be confounded with them.

Fractures, dislocations, malformations, etc., necrosis and caries of bones, Pott's disease, scoliosis, etc., are easy of X-ray diagnosis; but this subject is more strictly surgical, and is not within the scope of this chapter.

Records.—In making records, the examiner may trust to his memory and at his leisure sketch his findings on paper, or fill in blank forms; or he may place over the fluoroscopic screen a thin sheet of glass or celluloid, on which the outlines may be drawn. In order to keep the localities in mind, a metal rod can be placed on the patient to mark the spine, and other opaque objects placed over the nipples. This assists greatly in fixing the topography of the parts. The pencil may be covered with lead, as suggested by Williams, so that, as it makes the tracings, it can be seen through the fluoroscope.

Markings may be made directly on the skin with a good pencil, and afterward transferred to diagrams. This is, perhaps, the preferable method, especially in examinations of the chest. Tracing-cloth may be placed over the skin-markings, and the outlines traced on it.

Occasionally, false interpretation or incorrect observation leads to serious errors in the results of X-ray work. Only recently the daily press told of a case in which a prominent citizen missed his false teeth during the night, and concluded that he had swallowed them. The X-ray investigation apparently indicated that the missing teeth were lodged in the œsophagus, and an unsuccessful operation for their removal was performed. Hardly had the surgeon completed his work, when the missing teeth were found in the patient's bed.

A surgeon lately operated for ureteral calculus, in which the X-ray had apparently confirmed the clinical diagnosis of stone, but in which stone was not found by the operator.

Many other errors of a similar character have proven that at times the findings of the X-ray are either faulty in themselves or capable of being misinterpreted. Both of these are not only possibilities, but great probabilities. Imagination plays a great part in the interpretation of the fluoroscopic or radiographic image by the unskillful or the careless. The surgeon is often called upon to interpret from the radiograph taken by some one else, and the radiographer is frequently obliged to take a radiogram of a part in which he must use his own judgment, which cannot always be the best, without the aid of the medical or surgical diagnostician. Under these circumstances, errors of interpretation are liable to occur. Except in the most obvious instances, it must, for some time yet, be recognized that the X-ray has a limited diagnostic value in common with any other means of diagnosis. The surgical or medical clinician who relies upon someone else to do his X-ray work for him must always run the risk of two things:

first, that the radiographer may not supply all the necessary details in his radiogram, and, second, that he himself may not be able to properly interpret the shadows placed before him in the radiogram. To illustrate this point, a diagram is taken of the head and face. To the physician generally, such a picture, seen for the first time, would be unintelligible. Even after considerable experience, there are shadow-details in a radiogram of this character which require the closest study to properly understand. Especially is this true if empyema, fractures, tumors, foreign bodies, etc., exist to complicate the outlines.

X-ray work requires the highest type of intelligence. As in the case of the first assistant to the surgeon, it is often an advantage, in an operation, for the surgeon to have an assistant who is a better surgeon, even, than is the operator himself. So with the worker in X-rays,—if he has all the qualifications which belong to the clinician in addition to those which more properly belong to him as a radiographer, it requires little effort to appreciate the fact that he is far better equipped for the work than if he were simply an electrician who knows how to manipulate an energized Crookes' tube.

Errors will frequently occur in the use of the X-ray; but, in spite of its limitations and shortcomings, the fluoroscope and radiogram have exposed to view hitherto hidden secrets; have assisted in a most wonderful manner in establishing a new standard of normal conditions and relationship in the living human body; have helped in the determination of the existence, character and location of diseased tissues and disturbed relations in parts, which, for effectiveness, no other known means has ever been able to approach; and for all this the X-ray has been rightfully exalted as the greatest contribution to medicine and surgery in the closing years of the nineteenth century.

The literature on the subject of the X-ray has become very extensive. Societies devoted to its study and development have been formed in numerous places; men have set themselves apart for its special practice; voluminous and exhaustive works have been written with the distinctive title of "X-ray in Medicine and Surgery;" the whole civilized world, lay and scientific, has a fairly adequate idea of the character, value and method of operation of the X-ray; human life has been saved by its help; suffering has been mitigated, deformity corrected, and the sum total of human happiness has been greatly added to by the remarkable discovery of Roentgen.

For the assistance of those who may wish to study the subject as presented by masters in the art, most excellent publications are herewith referred to as perhaps the most thoroughly representative of the art:

"The Roentgen Rays in Medicine and Surgery."—Williams.
"The X-ray in Medicine and Surgery."—Walsh.
"The X-ray in Surgery."—Beck.
Archives of the Roentgen Ray (a journal).—London, England.
"Electricity in Medicine and Surgery" (chapter on the X-ray).—King.

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